```
=> s l1 and l3

L9          598 L1 AND L3

=> s l1 and l4

L10         501 L1 AND L4

=> s l1 and l5

L11         495 L1 AND L5

=> s l1 and l6

L12        1652 L1 AND L6

=> s l1 and l7

L13         632 L1 AND L7

=> file stnguide

COST IN U.S. DOLLARS                                SINCE FILE      TOTAL
                                                       ENTRY    SESSION
FULL ESTIMATED COST                                     2.60       4.49

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LAST RELOADED: Jul 20, 2007 (20070720/UP).

=> file hcaplus
COST IN U.S. DOLLARS                                SINCE FILE      TOTAL
                                                       ENTRY    SESSION
FULL ESTIMATED COST                                     0.06       4.55

FILE 'HCAPLUS' ENTERED AT 09:48:47 ON 26 JUL 2007
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FILE COVERS 1907 - 26 Jul 2007  VOL 147 ISS 5
FILE LAST UPDATED: 25 Jul 2007  (20070725/ED)

New CAS Information Use Policies, enter HELP USAGETERMS for details.

 This file contains CAS Registry Numbers for easy and accurate
 substance identification.

=> s l8 and l9 and l10
```

```
=> file hcaplus
COST IN U.S. DOLLARS                                    SINCE FILE      TOTAL
                                                           ENTRY    SESSION
FULL ESTIMATED COST                                         1.89       1.89

FILE 'HCAPLUS' ENTERED AT 09:48:06 ON 26 JUL 2007
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FILE COVERS 1907 - 26 Jul 2007  VOL 147 ISS 5
FILE LAST UPDATED: 25 Jul 2007  (20070725/ED)

New CAS Information Use Policies, enter HELP USAGETERMS for details.

 This file contains CAS Registry Numbers for easy and accurate
 substance identification.

=> s monoclonal(w)antibody

        147736 MONOCLONAL
        314233 ANTIBODY
L1       80206 MONOCLONAL(W)ANTIBODY

=> s GD3

L2        7336 GD3

=> s EGFR

L3        8897 EGFR

=> s HER2

L4        3727 HER2

=> s neuroblastoma

L5       16764 NEUROBLASTOMA

=> s melanoma

L6       35305 MELANOMA

=> s lymphoma(2a)Hodgkin

         38338 LYMPHOMA
         11330 HODGKIN
L7        7255 LYMPHOMA(2A)HODGKIN

=> s l1 and l2

L8         254 L1 AND L2
```

antibodies include polyclonal and monoclonal antibodies. NNV and IPNV are produced in an immortal cell line (GF-1) derived from the grouper fish E. coioides fin tissue, ATCC deposit number PTA-859. The present invention also provides methods for detecting viral infections in fish using enzyme immunoassay (EIA).

AN 2002:833302 HCAPLUS <<LOGINID::20070529>>
DN 137:351509
TI Immortal cell line derived from the grouper Epinephelus coioides and the applications thereof
IN Chi, Shau-Chi
PA Taiwan
SO U.S. Pat. Appl. Publ., 20 pp., Cont.-in-part of U.S. 6,436,702.
CODEN: USXXCO
DT Patent
LA English
FAN.CNT 4

	PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI	US 2002159993	A1	20021031	US 2001-4414	20011206 <--
	US 6436702	B1	20020820	US 1999-450696	19991130 <--
	US 2002164787	A1	20021107	US 2001-998212	20011203 <--
	US 6566117	B2	20030520		
PRAI	US 1998-110699P	P	19981203 <--		
	US 1999-450696	A2	19991130 <--		

L16 ANSWER 3 OF 3 HCAPLUS COPYRIGHT 2007 ACS on STN
TI Treatment of fungal infections with polyene or beta glucan synthase inhibitor antifungals combined with anti HSP90 antibodies
AB The present invention relates to novel compns. and prepns. that are effective antifungal agents, and a novel antibody which can be incorporated into the compns. and prepns.
AN 2001:762848 HCAPLUS <<LOGINID::20070529>>
DN 135:315585
TI Treatment of fungal infections with polyene or beta glucan synthase inhibitor antifungals combined with anti HSP90 antibodies
IN Burnie, James Peter
PA Neutec Pharma PLC, UK
SO PCT Int. Appl., 50 pp.
CODEN: PIXXD2
DT Patent
LA English
FAN.CNT 1

	PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI	WO 2001076627	A1	20011018	WO 2001-GB1195	20010320 <--
	W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZW				
	RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG				
	CA 2401836	A1	20011018	CA 2001-2401836	20010320 <--
	EP 1267925	A1	20030102	EP 2001-911971	20010320 <--
	R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL, TR				
	BR 2001009846	A	20030603	BR 2001-9846	20010320 <--
	JP 2003530357	T	20031014	JP 2001-574143	20010320 <--
	NZ 520899	A	20050324	NZ 2001-520899	20010320 <--
	RU 2262952	C2	20051027	RU 2002-129510	20010320 <--

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     FILE 'HCAPLUS' ENTERED AT 09:48:06 ON 26 JUL 2007
L1        80206 S MONOCLONAL(W)ANTIBODY
L2         7336 S GD3
L3         8897 S EGFR
L4         3727 S HER2
L5        16764 S NEUROBLASTOMA
L6        35305 S MELANOMA
L7         7255 S LYMPHOMA(2A)HODGKIN
L8          254 S L1 AND L2
L9          598 S L1 AND L3
L10         501 S L1 AND L4
L11         495 S L1 AND L5
L12        1652 S L1 AND L6
L13         632 S L1 AND L7

     FILE 'STNGUIDE' ENTERED AT 09:48:18 ON 26 JUL 2007

     FILE 'HCAPLUS' ENTERED AT 09:48:47 ON 26 JUL 2007
L14           0 S L8 AND L9 AND L10
L15          10 S L11 AND L12 AND L13

     FILE 'STNGUIDE' ENTERED AT 09:48:50 ON 26 JUL 2007

     FILE 'HCAPLUS' ENTERED AT 09:49:09 ON 26 JUL 2007
L16           2 S L8 AND L9
L17           0 S L8 AND L10
L18          57 S L9 AND L10

     FILE 'STNGUIDE' ENTERED AT 09:49:12 ON 26 JUL 2007

     FILE 'HCAPLUS' ENTERED AT 09:50:37 ON 26 JUL 2007
L19           4 S L15 AND (AY<2002 OR PY<2002 OR PRY<2002)
L20          10 S L18 AND (AY<2002 OR PY<2002 OR PRY<2002)

     FILE 'HCAPLUS' ENTERED AT 10:02:01 ON 26 JUL 2007
L21         351 S L11 AND (PY<2000 OR AY<2000 OR PRY<2000)
L22        1186 S L12 AND (PY<2000 OR AY<2000 OR PRY<2000)
L23         156 S L13 AND (PY<2000 OR AY<2000 OR PRY<2000)

     FILE 'STNGUIDE' ENTERED AT 10:02:12 ON 26 JUL 2007

     FILE 'HCAPLUS' ENTERED AT 10:03:52 ON 26 JUL 2007
L24         199 S L8 AND (PY<2000 OR AY<2000 OR PRY<2000)
L25         157 S L9 AND (PY<2000 OR AY<2000 OR PRY<2000)
L26          74 S L21 AND L22
L27           3 S L21 AND L23
L28           5 S L22 AND L23

     FILE 'STNGUIDE' ENTERED AT 10:04:02 ON 26 JUL 2007

     FILE 'HCAPLUS' ENTERED AT 10:05:01 ON 26 JUL 2007
L29           0 S L24 AND COMPLIMENT
L30           0 S L25 AND COMPLIMENT

     FILE 'STNGUIDE' ENTERED AT 10:05:03 ON 26 JUL 2007

     FILE 'HCAPLUS' ENTERED AT 10:05:16 ON 26 JUL 2007
L31          34 S L24 AND COMPLEMENT
L32           2 S L25 AND COMPLEMENT

     FILE 'HCAPLUS' ENTERED AT 10:18:24 ON 26 JUL 2007
L33          36 S L21 AND COMPLEMENT
L34           9 S L23 AND COMPLEMENT
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                                                        ENTRY     SESSION
FULL ESTIMATED COST                                      2.60        9.81

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=> d file hcaplus
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PY<2002 or PRY<2002)

YOU HAVE REQUESTED DATA FROM FILE 'HCAPLUS' - CONTINUE? (Y)/N:s l18 and (AY<2002 or
PY<2002 or PRY<2002)

YOU HAVE REQUESTED DATA FROM FILE 'HCAPLUS' - CONTINUE? (Y)/N:file stnguide

YOU HAVE REQUESTED DATA FROM FILE 'HCAPLUS' - CONTINUE? (Y)/N:n


=> file hcaplus

COST IN U.S. DOLLARS                                SINCE FILE      TOTAL
                                                         ENTRY    SESSION
FULL ESTIMATED COST                                       0.12       9.93

FILE 'HCAPLUS' ENTERED AT 09:50:37 ON 26 JUL 2007
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FILE COVERS 1907 - 26 Jul 2007  VOL 147 ISS 5
FILE LAST UPDATED: 25 Jul 2007  (20070725/ED)

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 This file contains CAS Registry Numbers for easy and accurate
 substance identification.

=> s l15 and (AY<2002 or PY<2002 or PRY<2002)

       4175881 AY<2002
      21894788 PY<2002
       3652712 PRY<2002
L19           4 L15 AND (AY<2002 OR PY<2002 OR PRY<2002)

=> s l18 and (AY<2002 or PY<2002 or PRY<2002)

       4175881 AY<2002
      21894788 PY<2002
       3652712 PRY<2002
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L20          10 L18 AND (AY<2002 OR PY<2002 OR PRY<2002)

=> file stnguide

COST IN U.S. DOLLARS                                SINCE FILE      TOTAL
                                                       ENTRY    SESSION
FULL ESTIMATED COST                                     2.60      12.53

FILE 'STNGUIDE' ENTERED AT 09:50:43 ON 26 JUL 2007
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FILE CONTAINS CURRENT INFORMATION.
LAST RELOADED: Jul 20, 2007 (20070720/UP).

=> d l19 104 ti abs bib
YOU HAVE REQUESTED DATA FROM FILE 'HCAPLUS' - CONTINUE? (Y)/N:y


     4 ANSWERS ARE AVAILABLE.  SPECIFIED ANSWER NUMBER EXCEEDS ANSWER SET SIZE
The answer numbers requested are not in the answer set.
ENTER ANSWER NUMBER OR RANGE (1):1-4

L19  ANSWER 1 OF 4  HCAPLUS  COPYRIGHT 2007 ACS on STN
TI   Methods and compositions for the prevention or treatment of neoplasia
     comprising a COX-2 inhibitor in combination with an epidermal growth
     factor receptor antagonist
AB   The present invention relates to a novel method of preventing and/or
     treating neoplasia disorders in a subject that is in need of such
     prevention or treatment by administering to the subject at least one COX-2
     inhibitor in combination with an EGF receptor antagonist.  Compns.,
     pharmaceutical compns. and kits are also described.
AN   2004:533970  HCAPLUS <<LOGINID::20070726>>
DN   141:65088
TI   Methods and compositions for the prevention or treatment of neoplasia
     comprising a COX-2 inhibitor in combination with an epidermal growth
     factor receptor antagonist
IN   Masferrer, Jaime
PA   Pharmacia Corporation, USA
SO   U.S. Pat. Appl. Publ., 103 pp., Cont.-in-part of U.S. Ser. No. 470,951.
     CODEN: USXXCO
DT   Patent
LA   English
FAN.CNT 21
     PATENT NO.          KIND   DATE        APPLICATION NO.          DATE
     --------------      ----   --------    --------------------     --------
PI   US 2004127470        A1    20040701    US 2003-651916           20030829 <--
     EP 1522313           A1    20050413    EP 2004-26577            19991222 <--
         R:  AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT,
             IE, FI, RO, CY
     WO 2005037259        A2    20050428    WO 2004-US27574          20040825
     WO 2005037259        A3    20050804
         W:  AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BW, BY, BZ, CA, CH,
             CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD,
             GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC,
             LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NA, NI,
             NO, NZ, OM, PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SY,
             TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW
         RW: BW, GH, GM, KE, LS, MW, MZ, NA, SD, SL, SZ, TZ, UG, ZM, ZW, AM,
             AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK,
             EE, ES, FI, FR, GB, GR, HU, IE, IT, LU, MC, NL, PL, PT, RO, SE,
             SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE,
             SN, TD, TG
     AU 2004210578        A1    20041007    AU 2004-210578           20040910 <--
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PRAI US 1998-113786P P 19981223 <--
US 1999-470951 B2 19991222 <--
US 1999-385214 A 19990827 <--
AU 2000-25936 A3 19991222 <--
EP 1999-968939 A3 19991222 <--
US 2003-651916 A 20030829

L19 ANSWER 2 OF 4 HCAPLUS COPYRIGHT 2007 ACS on STN
TI Direct targeting binding multivalent monospecific proteins of human
AB The present invention relates to multivalent, monospecific binding proteins. These binding proteins comprise two or more binding sites, where each binding site specifically binds to the same type of target cell, and preferably with the same antigen on such a target cell. The present invention further relates to compns. of monospecific diabodies, triabodies, and tetrabodies, and to recombinant vectors useful for the expression of these functional binding proteins in a microbial host. Also provided are methods of using invention compns. in the treatment and/or diagnosis of tumors.
AN 2003:320021 HCAPLUS <<LOGINID::20070726>>
DN 138:336427
TI Direct targeting binding multivalent monospecific proteins of human
IN Rossi, Edmund; Chang, Chien-Hsing Ken; Goldenberg, David M.
PA IBC Pharmaceuticals, USA; Immunomedics Inc.
SO PCT Int. Appl., 62 pp.
CODEN: PIXXD2
DT Patent
LA English
FAN.CNT 2

	PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI	WO 2003033654	A2	20030424	WO 2002-US32718	20021015 <--
	WO 2003033654	A3	20031113		
	W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, OM, PH, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW				
	RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG				
	CA 2463672	A1	20030424	CA 2002-2463672	20021015 <--
	AU 2002348437	A1	20030428	AU 2002-348437	20021015 <--
	US 2003148409	A1	20030807	US 2002-270073	20021015 <--
	EP 1448780	A2	20040825	EP 2002-782156	20021015 <--
	R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL, TR, BG, CZ, EE, SK				
	JP 2005507659	T	20050324	JP 2003-536384	20021015 <--
	CN 1604966	A	20050406	CN 2002-825068	20021015 <--
	BR 2002013303	A	20050607	BR 2002-13303	20021015 <--
	MX 2004PA03535	A	20050620	MX 2004-PA3535	20040415 <--
	IN 2004CN01047	A	20060203	IN 2004-CN1047	20040513 <--
PRAI	US 2001-328835P	P	20011015 <--		
	US 2001-341881P	P	20011221 <--		
	US 2002-345641P	P	20020108		
	US 2002-404919P	P	20020822		
	WO 2002-US32718	W	20021015		

L19 ANSWER 3 OF 4 HCAPLUS COPYRIGHT 2007 ACS on STN
TI Human glandular kallikrein (hK2)-specific monoclonal antibodies for diagnosis and treatment of hK2-expressing cancer
AB Disclosed are monoclonal antibodies preferentially binding hK2 over PSA. The monoclonal antibodies are generated by immunization with recombinant

hK2 producing in virus, bacteria, parasite, or tumor cells. The antibodies (6B7, 3E6, 1F8, 3C7, 11C4 and 9B4) are belong to IgG1, IgG2a, IgG2b, IgG2, IgG3 or IgG4 isotypes. These monoclonal antibodies are useful for immunodiagnosis of cancer, especially prostate cancer, in mammal such as mouse or human.

AN 2003:282716 HCAPLUS <<LOGINID::20070726>>
DN 138:302650
TI Human glandular kallikrein (hK2)-specific monoclonal antibodies for diagnosis and treatment of hK2-expressing cancer
IN Frelinger, John G.; Fisher, Terrence L.; Nocera, Mary Ann; Lord, Edith M.
PA University of Rochester, USA
SO PCT Int. Appl., 124 pp.
CODEN: PIXXD2
DT Patent
LA English
FAN.CNT 1

	PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI	WO 2003029427	A2	20030410	WO 2002-US31477	20021003 <--
	WO 2003029427	A3	20031218		
	W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, OM, PH, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW				
	RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG				
	AU 2002362447	A1	20030414	AU 2002-362447	20021003 <--
	US 2004219163	A1	20041104	US 2004-491761	20040527 <--
PRAI	US 2001-326772P	P	20011003	<--	
	WO 2002-US31477	W	20021003		

L19 ANSWER 4 OF 4 HCAPLUS COPYRIGHT 2007 ACS on STN
TI Method for relieving pain associated with an internal disease site
AB Methods are provided for in vivo administration of a pain-relieving drug, such as a local anesthetic (e.g. lidocaine), to an interior disease site for pain relief at the interior disease site. In the invention pain treatment methods, a subject is administered a targeting construct comprising a biol. compatible pain-relieving agent and a tumor-avid ligand or monoclonal antibody that preponderantly binds to or is taken up by the tissue associated with an interior disease site. Administration is by a method other than topical injection or application, such as parenteral injection. Because the pain-relieving agent is delivered by the ligand to the disease site, intractable pain situated in the interior of the body, such as is caused by various tumors, can be managed using a lower level of the pain-relieving agent then is required when the pain-relieving agent is injected in the free state.
AN 2001:489224 HCAPLUS <<LOGINID::20070726>>
DN 135:97445
TI Method for relieving pain associated with an internal disease site
IN Luiken, George A.
PA Fluoro Probe, Inc., USA
SO PCT Int. Appl., 31 pp.
CODEN: PIXXD2
DT Patent
LA English
FAN.CNT 1

	PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI	WO 2001047512	A2	20010705	WO 2000-US42661	20001206 <--

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     WO 2001047512         A3    20020502
         W:  AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN,
             CR, CU, CZ, DE, DK, DM, DZ, EE, ES, FI, GB, GD, GE, GH, GM, HR,
             HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT,
             LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, PL, PT, RO, RU,
             SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, US, UZ, VN,
             YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM
         RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY,
             DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR, BF,
             BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG
     AU 2001049041         A5    20010709    AU 2001-49041          20001206 <--
PRAI US 1999-457498        A1    19991208  <--
     WO 2000-US42661       W     20001206  <--


=> d l20 1-10 ti
YOU HAVE REQUESTED DATA FROM FILE 'HCAPLUS' - CONTINUE? (Y)/N:y


L20  ANSWER 1 OF 10  HCAPLUS  COPYRIGHT 2007 ACS on STN
TI   Antibody fragment-polymer conjugates with improved half-life, mean
     residence time, and/or clearance rate in circulation for disease diagnosis
     and therapy

L20  ANSWER 2 OF 10  HCAPLUS  COPYRIGHT 2007 ACS on STN
TI   Antibody fragment conjugated with polymer to improve half-life in
     circulation for diagnosis and therapy

L20  ANSWER 3 OF 10  HCAPLUS  COPYRIGHT 2007 ACS on STN
TI   Trastuzumab in the treatment of HER2 positive breast cancer

L20  ANSWER 4 OF 10  HCAPLUS  COPYRIGHT 2007 ACS on STN
TI   Epidermal growth factor receptor (HER1) tyrosine kinase inhibitor ZD1839
     (Iressa) inhibits HER2/neu (erbB2)-overexpressing breast cancer
     cells in vitro and in vivo

L20  ANSWER 5 OF 10  HCAPLUS  COPYRIGHT 2007 ACS on STN
TI   Non-radioisotopic method for the in vitro measurement of EGF receptor
     tyrosine kinase

L20  ANSWER 6 OF 10  HCAPLUS  COPYRIGHT 2007 ACS on STN
TI   Growth factors regulate heterogeneous nuclear ribonucleoprotein K
     expression and function

L20  ANSWER 7 OF 10  HCAPLUS  COPYRIGHT 2007 ACS on STN
TI   New perspectives on anti-HER2/neu therapeutics

L20  ANSWER 8 OF 10  HCAPLUS  COPYRIGHT 2007 ACS on STN
TI   Activation-dependent clustering of the erbB2 receptor tyrosine kinase
     detected by scanning near-field optical microscopy

L20  ANSWER 9 OF 10  HCAPLUS  COPYRIGHT 2007 ACS on STN
TI   Augmentation of a humanized anti-HER2 mAb 4D5 induced growth
     inhibition by a human-mouse chimeric anti-EGF receptor mAb C225

L20  ANSWER 10 OF 10  HCAPLUS  COPYRIGHT 2007 ACS on STN
TI   Clinical experience with CD64-directed immunotherapy. An overview


=> d l20 2 3 4 6 7 8 9 10
YOU HAVE REQUESTED DATA FROM FILE 'HCAPLUS' - CONTINUE? (Y)/N:y
```

L20 ANSWER 2 OF 10 HCAPLUS COPYRIGHT 2007 ACS on STN
AN 2006:367102 HCAPLUS <<LOGINID::20070726>>
DN 144:410813
TI Antibody fragment conjugated with polymer to improve half-life in circulation for diagnosis and therapy
IN Hsei, Vanessa; Koumenis, Iphigenia; Leong, Steven; Shahrokh, Zahra; Zapata, Gerardo
PA Genentech, Inc., USA
SO U.S. Pat. Appl. Publ., 283 pp., Cont. U.S. Ser. No. 489,394.
CODEN: USXXCO
DT Patent
LA English
FAN.CNT 6

	PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI	US 2006083683	A1	20060420	US 2005-259232	20051025 <--
	US 7214776	B2	20070508		
	US 7122636	B1	20061017	US 2000-489394	20000121 <--
	AU 2002300457	A1	20030213	AU 2002-300457	20020802 <--
	US 2007048219	A1	20070301	US 2006-541145	20060928 <--
PRAI	US 1999-116787P	P	19990121 <--		
	US 2000-489394	A1	20000121 <--		
	US 1997-38664P	P	19970221 <--		
	US 1997-804444	A2	19970221 <--		
	US 1998-12116	B2	19980122 <--		
	US 1998-74330P	P	19980122 <--		
	AU 1998-65357	A3	19980220 <--		
	US 1998-26985	A2	19980220 <--		
	US 1999-355014	A2	19990913 <--		
	US 2005-259232	A1	20051025		

RE.CNT 136 THERE ARE 136 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L20 ANSWER 3 OF 10 HCAPLUS COPYRIGHT 2007 ACS on STN
AN 2002:429802 HCAPLUS <<LOGINID::20070726>>
DN 137:41199
TI Trastuzumab in the treatment of HER2 positive breast cancer
AU Summerhayes, Maxwell
CS The Pharmacy Department, Guy's Hospital, London, SE1 9RT, UK
SO Journal of Oncology Pharmacy Practice (2001), 7(1), 9-25
CODEN: JOPPFI; ISSN: 1078-1552
PB Arnold, Hodder Headline
DT Journal; General Review
LA English
RE.CNT 66 THERE ARE 66 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L20 ANSWER 4 OF 10 HCAPLUS COPYRIGHT 2007 ACS on STN
AN 2002:12764 HCAPLUS <<LOGINID::20070726>>
DN 136:288673
TI Epidermal growth factor receptor (HER1) tyrosine kinase inhibitor ZD1839 (Iressa) inhibits HER2/neu (erbB2)-overexpressing breast cancer cells in vitro and in vivo
AU Moulder, Stacy L.; Yakes, F. Michael; Muthuswamy, Senthil K.; Bianco, Roberto; Simpson, Jean F.; Arteaga, Carlos L.
CS Department of Medicine, University School of Medicine, Nashville, TN, 37232-6307, USA
SO Cancer Research (2001), 61(24), 8887-8895
CODEN: CNREA8; ISSN: 0008-5472
PB American Association for Cancer Research
DT Journal
LA English
RE.CNT 51 THERE ARE 51 CITED REFERENCES AVAILABLE FOR THIS RECORD

ALL CITATIONS AVAILABLE IN THE RE FORMAT

L20 ANSWER 6 OF 10 HCAPLUS COPYRIGHT 2007 ACS on STN
AN 2001:267681 HCAPLUS <<LOGINID::20070726>>
DN 134:305768
TI Growth factors regulate heterogeneous nuclear ribonucleoprotein K expression and function
AU Mandal, Mahitosh; Vadlamudi, Ratna; Nguyen, Diep; Wang, Rui-An; Costa, Luis; Bagheri-Yarmand, Rozita; Mendelsohn, John; Kumar, Rakesh
CS Department of Molecular and Cellular Oncology, The University of Texas M. D. Anderson Cancer Center-108, Houston, TX, 77030, USA
SO Journal of Biological Chemistry (2001), 276(13), 9699-9704
CODEN: JBCHA3; ISSN: 0021-9258
PB American Society for Biochemistry and Molecular Biology
DT Journal
LA English
RE.CNT 44 THERE ARE 44 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L20 ANSWER 7 OF 10 HCAPLUS COPYRIGHT 2007 ACS on STN
AN 2000:856232 HCAPLUS <<LOGINID::20070726>>
DN 135:13714
TI New perspectives on anti-HER2/neu therapeutics
AU Zhang, Hong-Tao; Wang, Qiang; Greene, Mark I.; Murali, Ramachandran
CS Dept. of Pathology and Laboratory of Medicine, University of Pennsylvania, Philadelphia, PA, 19104, USA
SO Drug News & Perspectives (2000), 13(6), 325-329
CODEN: DNPEED; ISSN: 0214-0934
PB Prous Science
DT Journal; General Review
LA English
RE.CNT 38 THERE ARE 38 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L20 ANSWER 8 OF 10 HCAPLUS COPYRIGHT 2007 ACS on STN
AN 1999:404123 HCAPLUS <<LOGINID::20070726>>
DN 131:168551
TI Activation-dependent clustering of the erbB2 receptor tyrosine kinase detected by scanning near-field optical microscopy
AU Nagy, Peter; Jenei, Attila; Kirsch, Achim K.; Szollosi, Janos; Damjanovich, Sandor; Jovin, Thomas M.
CS Department of Molecular Biology, Max Planck Institute for Biophysical Chemistry, Gottingen, D-37077, Germany
SO Journal of Cell Science (1999), 112(11), 1733-1741
CODEN: JNCSAI; ISSN: 0021-9533
PB Company of Biologists Ltd.
DT Journal
LA English
RE.CNT 47 THERE ARE 47 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L20 ANSWER 9 OF 10 HCAPLUS COPYRIGHT 2007 ACS on STN
AN 1999:123040 HCAPLUS <<LOGINID::20070726>>
DN 130:310394
TI Augmentation of a humanized anti-HER2 mAb 4D5 induced growth inhibition by a human-mouse chimeric anti-EGF receptor mAb C225
AU Ye, Dingwei; Mendelsohn, John; Fan, Zhen
CS M.D. Anderson Cancer Center, The University of Texas, Houston, TX, 77030-4009, USA
SO Oncogene (1999), 18(3), 731-738
CODEN: ONCNES; ISSN: 0950-9232
PB Stockton Press
DT Journal
LA English

RE.CNT 39 THERE ARE 39 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L20 ANSWER 10 OF 10 HCAPLUS COPYRIGHT 2007 ACS on STN
AN 1998:23257 HCAPLUS <<LOGINID::20070726>>
DN 128:165944
TI Clinical experience with CD64-directed immunotherapy. An overview
AU Curnow, Randall T.
CS Medarex Inc., Annadale, NJ, 08801, USA
SO Cancer Immunology Immunotherapy (1997), 45(3/4), 210-215
CODEN: CIIMDN; ISSN: 0340-7004
PB Springer-Verlag
DT Journal; General Review
LA English
RE.CNT 9 THERE ARE 9 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

=> d l19 1-4 ti
YOU HAVE REQUESTED DATA FROM FILE 'HCAPLUS' - CONTINUE? (Y)/N:y

L19 ANSWER 1 OF 4 HCAPLUS COPYRIGHT 2007 ACS on STN
TI Methods and compositions for the prevention or treatment of neoplasia comprising a COX-2 inhibitor in combination with an epidermal growth factor receptor antagonist

L19 ANSWER 2 OF 4 HCAPLUS COPYRIGHT 2007 ACS on STN
TI Direct targeting binding multivalent monospecific proteins of human

L19 ANSWER 3 OF 4 HCAPLUS COPYRIGHT 2007 ACS on STN
TI Human glandular kallikrein (hK2)-specific monoclonal antibodies for diagnosis and treatment of hK2-expressing cancer

L19 ANSWER 4 OF 4 HCAPLUS COPYRIGHT 2007 ACS on STN
TI Method for relieving pain associated with an internal disease site

=> d l19 1-4 ti abs bib
YOU HAVE REQUESTED DATA FROM FILE 'HCAPLUS' - CONTINUE? (Y)/N:y

L19 ANSWER 1 OF 4 HCAPLUS COPYRIGHT 2007 ACS on STN
TI Methods and compositions for the prevention or treatment of neoplasia comprising a COX-2 inhibitor in combination with an epidermal growth factor receptor antagonist
AB The present invention relates to a novel method of preventing and/or treating neoplasia disorders in a subject that is in need of such prevention or treatment by administering to the subject at least one COX-2 inhibitor in combination with an EGF receptor antagonist. Compns., pharmaceutical compns. and kits are also described.
AN 2004:533970 HCAPLUS <<LOGINID::20070726>>
DN 141:65088
TI Methods and compositions for the prevention or treatment of neoplasia comprising a COX-2 inhibitor in combination with an epidermal growth factor receptor antagonist
IN Masferrer, Jaime
PA Pharmacia Corporation, USA
SO U.S. Pat. Appl. Publ., 103 pp., Cont.-in-part of U.S. Ser. No. 470,951.
CODEN: USXXCO
DT Patent
LA English

FAN.CNT 21

	PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI	US 2004127470	A1	20040701	US 2003-651916	20030829 <--
	EP 1522313	A1	20050413	EP 2004-26577	19991222 <--
	R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, FI, RO, CY				
	WO 2005037259	A2	20050428	WO 2004-US27574	20040825
	WO 2005037259	A3	20050804		
	W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BW, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NA, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW				
	RW: BW, GH, GM, KE, LS, MW, MZ, NA, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IT, LU, MC, NL, PL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG				
	AU 2004210578	A1	20041007	AU 2004-210578	20040910 <--
PRAI	US 1998-113786P	P	19981223 <--		
	US 1999-470951	B2	19991222 <--		
	US 1999-385214	A	19990827 <--		
	AU 2000-25936	A3	19991222 <--		
	EP 1999-968939	A3	19991222 <--		
	US 2003-651916	A	20030829		

L19 ANSWER 2 OF 4 HCAPLUS COPYRIGHT 2007 ACS on STN

TI Direct targeting binding multivalent monospecific proteins of human

AB The present invention relates to multivalent, monospecific binding proteins. These binding proteins comprise two or more binding sites, where each binding site specifically binds to the same type of target cell, and preferably with the same antigen on such a target cell. The present invention further relates to compns. of monospecific diabodies, triabodies, and tetrabodies, and to recombinant vectors useful for the expression of these functional binding proteins in a microbial host. Also provided are methods of using invention compns. in the treatment and/or diagnosis of tumors.

AN 2003:320021 HCAPLUS <<LOGINID::20070726>>

DN 138:336427

TI Direct targeting binding multivalent monospecific proteins of human

IN Rossi, Edmund; Chang, Chien-Hsing Ken; Goldenberg, David M.

PA IBC Pharmaceuticals, USA; Immunomedics Inc.

SO PCT Int. Appl., 62 pp.
CODEN: PIXXD2

DT Patent

LA English

FAN.CNT 2

	PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI	WO 2003033654	A2	20030424	WO 2002-US32718	20021015 <--
	WO 2003033654	A3	20031113		
	W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, OM, PH, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW				
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	CA 2463672	A1	20030424	CA 2002-2463672	20021015 <--

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     AU 2002348437         A1    20030428    AU 2002-348437         20021015 <--
     US 2003148409         A1    20030807    US 2002-270073         20021015 <--
     EP 1448780            A2    20040825    EP 2002-782156         20021015 <--
         R:  AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT,
             IE, SI, LT, LV, FI, RO, MK, CY, AL, TR, BG, CZ, EE, SK
     JP 2005507659         T     20050324    JP 2003-536384         20021015 <--
     CN 1604966            A     20050406    CN 2002-825068         20021015 <--
     BR 2002013303         A     20050607    BR 2002-13303          20021015 <--
     MX 2004PA03535        A     20050620    MX 2004-PA3535         20040415 <--
     IN 2004CN01047        A     20060203    IN 2004-CN1047         20040513 <--
PRAI US 2001-328835P       P     20011015  <--
     US 2001-341881P       P     20011221  <--
     US 2002-345641P       P     20020108
     US 2002-404919P       P     20020822
     WO 2002-US32718       W     20021015

L19  ANSWER 3 OF 4  HCAPLUS  COPYRIGHT 2007 ACS on STN
TI   Human glandular kallikrein (hK2)-specific monoclonal antibodies for
     diagnosis and treatment of hK2-expressing cancer
AB   Disclosed are monoclonal antibodies preferentially binding hK2 over PSA.
     The monoclonal antibodies are generated by immunization with recombinant
     hK2 producing in virus, bacteria, parasite, or tumor cells.  The
     antibodies (6B7, 3E6, 1F8, 3C7, 11C4 and 9B4) are belong to IgG1, IgG2a,
     IgG2b, IgG2, IgG3 or IgG4 isotypes.  These monoclonal antibodies are
     useful for immunodiagnosis of cancer, especially prostate cancer, in mammal
such
     as mouse or human.
AN   2003:282716  HCAPLUS <<LOGINID::20070726>>
DN   138:302650
TI   Human glandular kallikrein (hK2)-specific monoclonal antibodies for
     diagnosis and treatment of hK2-expressing cancer
IN   Frelinger, John G.; Fisher, Terrence L.; Nocera, Mary Ann; Lord, Edith M.
PA   University of Rochester, USA
SO   PCT Int. Appl., 124 pp.
     CODEN: PIXXD2
DT   Patent
LA   English
FAN.CNT 1
     PATENT NO.            KIND   DATE        APPLICATION NO.        DATE
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PI   WO 2003029427          A2    20030410    WO 2002-US31477        20021003 <--
     WO 2003029427          A3    20031218
         W:  AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN,
             CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH,
             GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR,
             LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, OM, PH,
             PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TN, TR, TT, TZ,
             UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW
         RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY,
             KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES,
             FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, SK, TR, BF, BJ, CF,
             CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG
     AU 2002362447          A1    20030414    AU 2002-362447         20021003 <--
     US 2004219163          A1    20041104    US 2004-491761         20040527 <--
PRAI US 2001-326772P        P     20011003  <--
     WO 2002-US31477        W     20021003

L19  ANSWER 4 OF 4  HCAPLUS  COPYRIGHT 2007 ACS on STN
TI   Method for relieving pain associated with an internal disease site
AB   Methods are provided for in vivo administration of a pain-relieving drug,
     such as a local anesthetic (e.g. lidocaine), to an interior disease site
     for pain relief at the interior disease site.  In the invention pain
     treatment methods, a subject is administered a targeting construct
     comprising a biol. compatible pain-relieving agent and a tumor-avid ligand
```

or monoclonal antibody that preponderantly binds to or
is taken up by the tissue associated with an interior disease site.
Administration is by a method other than topical injection or application,
such as parenteral injection. Because the pain-relieving agent is
delivered by the ligand to the disease site, intractable pain situated in
the interior of the body, such as is caused by various tumors, can be
managed using a lower level of the pain-relieving agent then is required
when the pain-relieving agent is injected in the free state.

AN 2001:489224 HCAPLUS <<LOGINID::20070726>>
DN 135:97445
TI Method for relieving pain associated with an internal disease site
IN Luiken, George A.
PA Fluoro Probe, Inc., USA
SO PCT Int. Appl., 31 pp.
CODEN: PIXXD2
DT Patent
LA English
FAN.CNT 1

	PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI	WO 2001047512	A2	20010705	WO 2000-US42661	20001206 <--
	WO 2001047512	A3	20020502		
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	RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG				
	AU 2001049041	A5	20010709	AU 2001-49041	20001206 <--
PRAI	US 1999-457498	A1	19991208	<--	
	WO 2000-US42661	W	20001206	<--	

=> d his

(FILE 'HOME' ENTERED AT 09:42:51 ON 26 JUL 2007)

FILE 'HCAPLUS' ENTERED AT 09:48:06 ON 26 JUL 2007

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L1         80206 S MONOCLONAL(W)ANTIBODY
L2          7336 S GD3
L3          8897 S EGFR
L4          3727 S HER2
L5         16764 S NEUROBLASTOMA
L6         35305 S MELANOMA
L7          7255 S LYMPHOMA(2A)HODGKIN
L8           254 S L1 AND L2
L9           598 S L1 AND L3
L10          501 S L1 AND L4
L11          495 S L1 AND L5
L12         1652 S L1 AND L6
L13          632 S L1 AND L7
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FILE 'STNGUIDE' ENTERED AT 09:48:18 ON 26 JUL 2007

FILE 'HCAPLUS' ENTERED AT 09:48:47 ON 26 JUL 2007

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L14             0 S L8 AND L9 AND L10
L15            10 S L11 AND L12 AND L13
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FILE 'STNGUIDE' ENTERED AT 09:48:50 ON 26 JUL 2007

FILE 'HCAPLUS' ENTERED AT 09:49:09 ON 26 JUL 2007

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L16             2 S L8 AND L9
L17             0 S L8 AND L10
L18            57 S L9 AND L10

     FILE 'STNGUIDE' ENTERED AT 09:49:12 ON 26 JUL 2007

     FILE 'HCAPLUS' ENTERED AT 09:50:37 ON 26 JUL 2007
L19             4 S L15 AND (AY<2002 OR PY<2002 OR PRY<2002)
L20            10 S L18 AND (AY<2002 OR PY<2002 OR PRY<2002)

     FILE 'STNGUIDE' ENTERED AT 09:50:43 ON 26 JUL 2007

     FILE 'HCAPLUS' ENTERED AT 09:50:58 ON 26 JUL 2007

     FILE 'STNGUIDE' ENTERED AT 09:51:07 ON 26 JUL 2007

     FILE 'HCAPLUS' ENTERED AT 09:51:40 ON 26 JUL 2007

     FILE 'STNGUIDE' ENTERED AT 09:51:41 ON 26 JUL 2007

     FILE 'HCAPLUS' ENTERED AT 09:52:21 ON 26 JUL 2007

     FILE 'STNGUIDE' ENTERED AT 09:52:22 ON 26 JUL 2007

     FILE 'HCAPLUS' ENTERED AT 09:52:33 ON 26 JUL 2007

     FILE 'STNGUIDE' ENTERED AT 09:52:33 ON 26 JUL 2007

     FILE 'HCAPLUS' ENTERED AT 09:52:45 ON 26 JUL 2007

     FILE 'STNGUIDE' ENTERED AT 09:52:46 ON 26 JUL 2007

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FULL ESTIMATED COST                                 0.06      62.73

DISCOUNT AMOUNTS (FOR QUALIFYING ACCOUNTS)      SINCE FILE      TOTAL
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CA SUBSCRIBER PRICE                                 0.00      -6.24

 SESSION WILL BE HELD FOR 120 MINUTES
STN INTERNATIONAL SESSION SUSPENDED AT 09:53:14 ON 26 JUL 2007

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LOGINID:SSPTAEXO1623

PASSWORD:
 * * * * * * RECONNECTED TO STN INTERNATIONAL * * * * * *
SESSION RESUMED IN FILE 'STNGUIDE' AT 10:01:08 ON 26 JUL 2007
FILE 'STNGUIDE' ENTERED AT 10:01:08 ON 26 JUL 2007
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COST IN U.S. DOLLARS                            SINCE FILE      TOTAL
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FULL ESTIMATED COST                                 0.06      62.73

DISCOUNT AMOUNTS (FOR QUALIFYING ACCOUNTS)      SINCE FILE      TOTAL
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CA SUBSCRIBER PRICE                                      0.00      -6.24

=> file hcaplus
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CA SUBSCRIBER PRICE                                      0.00      -6.24

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PLEASE SEE "HELP USAGETERMS" FOR DETAILS.
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FILE COVERS 1907 - 26 Jul 2007  VOL 147 ISS 5
FILE LAST UPDATED: 25 Jul 2007  (20070725/ED)

New CAS Information Use Policies, enter HELP USAGETERMS for details.

 This file contains CAS Registry Numbers for easy and accurate
 substance identification.

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       3666867 AY<2000
       3139881 PRY<2000
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       3666867 AY<2000
       3139881 PRY<2000
L23         156 L13 AND (PY<2000 OR AY<2000 OR PRY<2000)

=> d l16 1-2 ti abs bib


L16  ANSWER 1 OF 2  HCAPLUS  COPYRIGHT 2007 ACS on STN
TI   Potent T cell modulating bispecific scFv constructs comprising modified
     VH-CDR3 region of anti-human CD3 antibody, OKT3, and therapeutic uses
     thereof
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AB In accordance with the present invention it was found that a CDR3 region of an antibody mol., preferably directed against the CD3 on the surface of a T-cell, may be specifically modified. This specific modification(s)/mutation(s) as disclosed herein provide for modified antibody constructs as disclosed herein with altered physiol. and/or biochem. activities. The invention describes the use of bispecific scFv constructs comprising anti-human EpCAM x anti-human CD3 for generation of mutants in the VH part of mouse anti-human CD3 monoclonal antibody OKT3. The present invention describes antibody construct comprising at least one CDR3 region, wherein comprises at least one substitution in the amino acid sequence YYDDHY (SEQ ID NO.1). This at least one substitution comprises: in the first position of SEQ ID NO.1 a substitution from Y to H; in the second position a substitution from Y to S, from Y to N, from Y to F or from Y to H; in third position a substitution from D to N or from D to E; in the forth position of a substitution from D to Q, from D to A, from D to V, from D to E or from D to G; in the fifth position a substitution from H to Q, from H to P, from H to Y, from H to R or from H to N; or in the sixth position a substitution from Y to N.
AN 2004:681433 HCAPLUS <<LOGINID::20070726>>
DN 141:205678
TI Potent T cell modulating bispecific scFv constructs comprising modified VH-CDR3 region of anti-human CD3 antibody, OKT3, and therapeutic uses thereof
IN Lanzavecchia, Antonio
PA Micromet AG, Germany
SO U.S. Pat. Appl. Publ., 95 pp.
CODEN: USXXCO
DT Patent
LA English
FAN.CNT 1

	PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI	US 2004162411	A1	20040819	US 2003-682845	20031010
	CA 2403313	A1	20040411	CA 2002-2403313	20021011
PRAI	CA 2002-2403313	A	20021011		
	US 2002-419149P	P	20021018		

L16 ANSWER 2 OF 2 HCAPLUS COPYRIGHT 2007 ACS on STN
TI Monoclonal antibodies to malignant human gliomas
AB Operationally specific monoclonal antibodies (MAbs) reactive with tumor but not normal adult tissues offer great potential for diagnosis and therapy of CNS neoplasms. Two targets for specific MAb localization were chosen for this study: (1) glioma-associated gangliosides GM2 [II3NeuAc-GgOse3Cer], GD2 [II3(NeuAc)2-GgOse3Cer], GD3 [II3(NeuAc)2-LacCer], 3'-isoLM1[IV3NeuAc-LcOse4Cer], and 3',6'-isoLD1 [IV3NeuAc,III6NeuAc-LcOSe4Cer] and (2) epidermal growth factor receptor (EGFR) variant mols. Epitopic specificity of isolated ganglioside hybridomas was determined with FAB-MS defined ganglioside stds. All MAb are IgM. Assay of 14 cytol. specimens and 31 frozen sections of primary CNS neoplasms revealed staining with anti-GD3 (14/14, 31/31), anti-GM2 (9/14, 26/31), and anti-GD2 (6/14, 24/30), resp. 3'-IsoLM1 and 3',6'-isoLD1, which exhibit a restricted oncofetal expression pattern and are not detectable in adult human brain, are present in 15/31 primary CNS neoplasms and in 1/8 human glioma xenografts, as detected by MAbs SL-50 and DMAb-14, resp. EGFR proteins, the second target, have unique amino acid spans resulting from gene deletion in the amplified EGFR gene present in subsets of malignant human gliomas. Antibodies against EGFR deletion-mutant Type III show highly restricted activity with a subset of glioma biopsies (6/35) expressing the mutant EGFR. These reagents should be useful for in vitro and in vivo diagnosis and, potentially, for treatment of malignant brain tumors.
AN 1993:144602 HCAPLUS <<LOGINID::20070726>>

DN 118:144602
TI Monoclonal antibodies to malignant human gliomas
AU Wikstrand, Carol J.; Fredman, Pam; Svennerholm, Lars; Humphrey, Peter A.; Bigner, Sandra H.; Bigner, Darell D.
CS Med. Cent., Duke Univ., Durham, NC, 27710, USA
SO Molecular and Chemical Neuropathology (1992), 17(2), 137-46
CODEN: MCHNEM; ISSN: 1044-7393
DT Journal
LA English

=> file stnguide

COST IN U.S. DOLLARS	SINCE FILE ENTRY	TOTAL SESSION
FULL ESTIMATED COST	8.26	71.05

DISCOUNT AMOUNTS (FOR QUALIFYING ACCOUNTS)	SINCE FILE ENTRY	TOTAL SESSION
CA SUBSCRIBER PRICE	-1.56	-7.80

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=> file hcaplus

COST IN U.S. DOLLARS	SINCE FILE ENTRY	TOTAL SESSION
FULL ESTIMATED COST	0.18	71.23

DISCOUNT AMOUNTS (FOR QUALIFYING ACCOUNTS)	SINCE FILE ENTRY	TOTAL SESSION
CA SUBSCRIBER PRICE	0.00	-7.80

FILE 'HCAPLUS' ENTERED AT 10:03:52 ON 26 JUL 2007
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FILE COVERS 1907 - 26 Jul 2007 VOL 147 ISS 5
FILE LAST UPDATED: 25 Jul 2007 (20070725/ED)

New CAS Information Use Policies, enter HELP USAGETERMS for details.

This file contains CAS Registry Numbers for easy and accurate substance identification.

=> s l8 and (PY<2000 or AY<2000 or PRY<2000)

20029282 PY<2000

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       3666867 AY<2000
       3139881 PRY<2000
L24         199 L8 AND (PY<2000 OR AY<2000 OR PRY<2000)

=> s l9 and (PY<2000 or AY<2000 or PRY<2000)

      20029282 PY<2000
       3666867 AY<2000
       3139881 PRY<2000
L25         157 L9 AND (PY<2000 OR AY<2000 OR PRY<2000)

=> s l21 and l22

L26          74 L21 AND L22

=> s l21 and l23

L27           3 L21 AND L23

=> s l22 and l23

L28           5 L22 AND L23

=> file stnguide

COST IN U.S. DOLLARS                                SINCE FILE      TOTAL
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FULL ESTIMATED COST                                    2.60      73.83

DISCOUNT AMOUNTS (FOR QUALIFYING ACCOUNTS)          SINCE FILE      TOTAL
                                                      ENTRY    SESSION
CA SUBSCRIBER PRICE                                    0.00      -7.80

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FILE CONTAINS CURRENT INFORMATION.
LAST RELOADED: Jul 20, 2007 (20070720/UP).

=> file hcaplus
COST IN U.S. DOLLARS                                SINCE FILE      TOTAL
                                                      ENTRY    SESSION
FULL ESTIMATED COST                                    0.12      73.95

DISCOUNT AMOUNTS (FOR QUALIFYING ACCOUNTS)          SINCE FILE      TOTAL
                                                      ENTRY    SESSION
CA SUBSCRIBER PRICE                                    0.00      -7.80

FILE 'HCAPLUS' ENTERED AT 10:05:01 ON 26 JUL 2007
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FILE COVERS 1907 - 26 Jul 2007 VOL 147 ISS 5
FILE LAST UPDATED: 25 Jul 2007 (20070725/ED)

New CAS Information Use Policies, enter HELP USAGETERMS for details.

This file contains CAS Registry Numbers for easy and accurate substance identification.

=> s l24 and compliment

          426 COMPLIMENT
L29   0 L24 AND COMPLIMENT

=> s l25 and compliment

          426 COMPLIMENT
L30   0 L25 AND COMPLIMENT

=> file stnguide

COST IN U.S. DOLLARS	SINCE FILE ENTRY	TOTAL SESSION
FULL ESTIMATED COST	2.60	76.55

DISCOUNT AMOUNTS (FOR QUALIFYING ACCOUNTS)	SINCE FILE ENTRY	TOTAL SESSION
CA SUBSCRIBER PRICE	0.00	-7.80

FILE 'STNGUIDE' ENTERED AT 10:05:03 ON 26 JUL 2007
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FILE CONTAINS CURRENT INFORMATION.
LAST RELOADED: Jul 20, 2007 (20070720/UP).

=> file hcaplus

COST IN U.S. DOLLARS	SINCE FILE ENTRY	TOTAL SESSION
FULL ESTIMATED COST	0.06	76.61

DISCOUNT AMOUNTS (FOR QUALIFYING ACCOUNTS)	SINCE FILE ENTRY	TOTAL SESSION
CA SUBSCRIBER PRICE	0.00	-7.80

FILE 'HCAPLUS' ENTERED AT 10:05:16 ON 26 JUL 2007
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FILE COVERS 1907 - 26 Jul 2007 VOL 147 ISS 5
FILE LAST UPDATED: 25 Jul 2007 (20070725/ED)

New CAS Information Use Policies, enter HELP USAGETERMS for details.

This file contains CAS Registry Numbers for easy and accurate substance identification.

=> s l24 and complement

```
         69689 COMPLEMENT
L31         34 L24 AND COMPLEMENT
```

=> s l25 and complement

```
         69689 COMPLEMENT
L32          2 L25 AND COMPLEMENT
```

=> file stnguide

COST IN U.S. DOLLARS	SINCE FILE ENTRY	TOTAL SESSION
FULL ESTIMATED COST	2.60	79.21

DISCOUNT AMOUNTS (FOR QUALIFYING ACCOUNTS)	SINCE FILE ENTRY	TOTAL SESSION
CA SUBSCRIBER PRICE	0.00	-7.80

FILE 'STNGUIDE' ENTERED AT 10:05:18 ON 26 JUL 2007
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=> file stnguide

COST IN U.S. DOLLARS	SINCE FILE ENTRY	TOTAL SESSION
FULL ESTIMATED COST	0.06	79.27

DISCOUNT AMOUNTS (FOR QUALIFYING ACCOUNTS)	SINCE FILE ENTRY	TOTAL SESSION
CA SUBSCRIBER PRICE	0.00	-7.80

FILE 'STNGUIDE' ENTERED AT 10:05:19 ON 26 JUL 2007
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FILE CONTAINS CURRENT INFORMATION.
LAST RELOADED: Jul 20, 2007 (20070720/UP).

=> file stnguide

COST IN U.S. DOLLARS	SINCE FILE ENTRY	TOTAL SESSION
FULL ESTIMATED COST	0.06	79.33

DISCOUNT AMOUNTS (FOR QUALIFYING ACCOUNTS)	SINCE FILE ENTRY	TOTAL SESSION
CA SUBSCRIBER PRICE	0.00	-7.80

FILE 'STNGUIDE' ENTERED AT 10:05:19 ON 26 JUL 2007
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FILE CONTAINS CURRENT INFORMATION.

LAST RELOADED: Jul 20, 2007 (20070720/UP).

=> file stnguide

COST IN U.S. DOLLARS	SINCE FILE ENTRY	TOTAL SESSION
FULL ESTIMATED COST	0.06	79.39

DISCOUNT AMOUNTS (FOR QUALIFYING ACCOUNTS)	SINCE FILE ENTRY	TOTAL SESSION
CA SUBSCRIBER PRICE	0.00	-7.80

FILE 'STNGUIDE' ENTERED AT 10:05:20 ON 26 JUL 2007
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FILE CONTAINS CURRENT INFORMATION.
LAST RELOADED: Jul 20, 2007 (20070720/UP).

=> d l31 -110 ti
YOU HAVE REQUESTED DATA FROM FILE 'HCAPLUS' - CONTINUE? (Y)/N:y

L31 ANSWER 1 OF 34 HCAPLUS COPYRIGHT 2007 ACS on STN
TI Monoclonal antibodies raised against Guillain-Barre syndrome-associated Campylobacter jejuni lipopolysaccharides react with neuronal gangliosides and paralyze muscle-nerve preparations. [Erratum to document cited in CA131:309736]

L31 ANSWER 2 OF 34 HCAPLUS COPYRIGHT 2007 ACS on STN
TI Immunogenicity of a fucosyl-GM1-keyhole limpet hemocyanin conjugate vaccine in patients with small cell lung cancer

L31 ANSWER 3 OF 34 HCAPLUS COPYRIGHT 2007 ACS on STN
TI Biologic roles of gangliosides GM3 and GD3 in the attachment of human melanoma cells to extracellular matrix proteins

L31 ANSWER 4 OF 34 HCAPLUS COPYRIGHT 2007 ACS on STN
TI Monoclonal antibodies raised against Guillain-Barre syndrome-associated Campylobacter jejuni lipopolysaccharides react with neuronal gangliosides and paralyze muscle-nerve preparations

L31 ANSWER 5 OF 34 HCAPLUS COPYRIGHT 2007 ACS on STN
TI Anti-melanoma effects of R24, a monoclonal antibody against GD3 ganglioside

L31 ANSWER 6 OF 34 HCAPLUS COPYRIGHT 2007 ACS on STN
TI Administration of R24 monoclonal antibody and low-dose interleukin 2 for malignant melanoma

L31 ANSWER 7 OF 34 HCAPLUS COPYRIGHT 2007 ACS on STN
TI Human antibodies derived from immunized xenomice

L31 ANSWER 8 OF 34 HCAPLUS COPYRIGHT 2007 ACS on STN
TI Human melanoma cell lines deficient in GD3 ganglioside expression exhibit altered growth and tumorigenic characteristics

L31 ANSWER 9 OF 34 HCAPLUS COPYRIGHT 2007 ACS on STN
TI Levels of cell membrane CD59 regulate the extent of complement -mediated lysis of human melanoma cells

L31 ANSWER 10 OF 34 HCAPLUS COPYRIGHT 2007 ACS on STN
TI Lysis of human tumor cell lines by canine complement plus

monoclonal antiganglioside antibodies or natural canine xenoantibodies

L31 ANSWER 11 OF 34 HCAPLUS COPYRIGHT 2007 ACS on STN
TI Mapping effector functions of a monoclonal antibody to GD3 by characterization of a mouse-human chimeric antibody

L31 ANSWER 12 OF 34 HCAPLUS COPYRIGHT 2007 ACS on STN
TI Chemotactic activity of substances derived from antibody-loaded tumor cells on granulocytes

L31 ANSWER 13 OF 34 HCAPLUS COPYRIGHT 2007 ACS on STN
TI Immunocytochemical study on internalization of anti-carbohydrate monoclonal antibodies

L31 ANSWER 14 OF 34 HCAPLUS COPYRIGHT 2007 ACS on STN
TI Cell surface reactive human monoclonal antibody directed to human melanoma-associated gangliosides

L31 ANSWER 15 OF 34 HCAPLUS COPYRIGHT 2007 ACS on STN
TI Targeted neutralization of the complement membrane attack complex inhibitor CD59 on the surface of human melanoma cells

L31 ANSWER 16 OF 34 HCAPLUS COPYRIGHT 2007 ACS on STN
TI Biotinylation of monoclonal antibodies prevents their ability to activate the classical pathway of complement

L31 ANSWER 17 OF 34 HCAPLUS COPYRIGHT 2007 ACS on STN
TI A mouse/human chimeric anti-(ganglioside GD3) antibody with enhanced antitumor activities

L31 ANSWER 18 OF 34 HCAPLUS COPYRIGHT 2007 ACS on STN
TI Antitumor effects of a novel monoclonal antibody with high binding affinity to ganglioside GD3

L31 ANSWER 19 OF 34 HCAPLUS COPYRIGHT 2007 ACS on STN
TI Sensitive detection of ganglioside GD3 on the cell surface using liposome immune lysis assay

L31 ANSWER 20 OF 34 HCAPLUS COPYRIGHT 2007 ACS on STN
TI Molecular basis of complement resistance of human melanoma cells expressing the C3-cleaving membrane protease p65

L31 ANSWER 21 OF 34 HCAPLUS COPYRIGHT 2007 ACS on STN
TI Immunorecognition of ganglioside epitopes: correlation between affinity and cytotoxicity of ganglioside antibodies

L31 ANSWER 22 OF 34 HCAPLUS COPYRIGHT 2007 ACS on STN
TI Monoclonal antibodies to glycolipid carbohydrate chains for antitumor agents

L31 ANSWER 23 OF 34 HCAPLUS COPYRIGHT 2007 ACS on STN
TI Production of monoclonal antibodies specific for ganglioside GD3

L31 ANSWER 24 OF 34 HCAPLUS COPYRIGHT 2007 ACS on STN
TI Monoclonal antibodies to disialogangliosides: characterization of antibody-mediated cytotoxicity against human melanoma and neuroblastoma cells in vitro

L31 ANSWER 25 OF 34 HCAPLUS COPYRIGHT 2007 ACS on STN
TI Light chain variants of an IgG3 anti-GD3 monoclonal antibody and the relationship among avidity, effector functions, tumor targeting, and antitumor activity

L31 ANSWER 26 OF 34 HCAPLUS COPYRIGHT 2007 ACS on STN

TI Tumor immunotherapy, immunoprophylaxis, and assays using antiidiotypic antibodies

L31 ANSWER 27 OF 34 HCAPLUS COPYRIGHT 2007 ACS on STN
TI New anti-GD2 monoclonal antibodies produced from gamma-interferon-treated neuroblastoma cells

L31 ANSWER 28 OF 34 HCAPLUS COPYRIGHT 2007 ACS on STN
TI Immune and nonimmune effector functions of IgG3 mouse monoclonal antibody R24 detecting the disialoganglioside GD3 on the surface of melanoma cells

L31 ANSWER 29 OF 34 HCAPLUS COPYRIGHT 2007 ACS on STN
TI Tumor therapy with biologically active anti-tumor antibodies

L31 ANSWER 30 OF 34 HCAPLUS COPYRIGHT 2007 ACS on STN
TI Monoclonal antibody-defined correlations in melanoma between levels of GD2 and GD3 antigens and antibody-mediated cytotoxicity

L31 ANSWER 31 OF 34 HCAPLUS COPYRIGHT 2007 ACS on STN
TI Biosynthesis and expression of the disialoganglioside GD2, a relevant target antigen on small cell lung carcinoma for monoclonal antibody-mediated cytolysis

L31 ANSWER 32 OF 34 HCAPLUS COPYRIGHT 2007 ACS on STN
TI A molecular mechanism of complement resistance of human melanoma cells

L31 ANSWER 33 OF 34 HCAPLUS COPYRIGHT 2007 ACS on STN
TI Disialoganglioside GD3 on human melanoma serves as a relevant target antigen for monoclonal antibody-mediated tumor cytolysis

L31 ANSWER 34 OF 34 HCAPLUS COPYRIGHT 2007 ACS on STN
TI Inhibition of human melanoma cell growth in vitro by monoclonal anti-GD3-ganglioside antibody

=> d l31 5 6 9 11 17 18 21 ti abs bib
YOU HAVE REQUESTED DATA FROM FILE 'HCAPLUS' - CONTINUE? (Y)/N:y

L31 ANSWER 5 OF 34 HCAPLUS COPYRIGHT 2007 ACS on STN
TI Anti-melanoma effects of R24, a monoclonal antibody against GD3 ganglioside
AB R24, a mouse monoclonal antibody against GD3 ganglioside, is potent at mediating in vitro effector functions such as human complement-mediated cytotoxicity and antibody-dependent cellular cytotoxicity, and can block melanoma tumor growth in animal models. Because of these properties and the fact that GD3 is abundantly expressed on virtually all melanomas but is found on few normal tissues, R24 has been tested in a series of clin. trials in patients with metastatic melanoma. As a single agent, R24 can induce responses in patients treated with metastatic melanoma. Overall, there have been 10 responders out of 103 patients reported; two responses have been complete responses. Responses have largely occurred in patients treated with intermediate doses of R24 and have included complete responses. Combining R24 with either cytotoxic drugs or cytokines has not increased this response rate, although one trial with R24 and interleukin-2 resulted in a 43% response rate and merits further investigation. Local-regional treatments R24 (intratumor injections, regional limb perfusion, intrathecal administration) have also been attempted in a small number of

patients and responses have been described. Taken together, multiple centers have reported responses in patients with metastatic melanoma treated with R24.

AN 1997:749125 HCAPLUS <<LOGINID::20070726>>
DN 128:33536
TI Anti-melanoma effects of R24, a monoclonal antibody against GD3 ganglioside
AU Nasi, M. Laura; Meyers, Michael; Livingston, Philip O.; Houghton, Alan N.; Chapman, Paul B.
CS Department of Medicine, Clinical Immunology Service, Memorial Sloan-Kettering Cancer Center and Cornell University Medical College, New York, NY, 10021, USA
SO Melanoma Research (1997), 7(Suppl. 2), S155-S162
CODEN: MREEEH; ISSN: 0960-8931
PB Rapid Science Publishers
DT Journal; General Review
LA English
RE.CNT 44 THERE ARE 44 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L31 ANSWER 6 OF 34 HCAPLUS COPYRIGHT 2007 ACS on STN
TI Administration of R24 monoclonal antibody and low-dose interleukin 2 for malignant melanoma
AB R24 is a monoclonal antibody that recognizes the disialoganglioside GD3 expressed on the surface of malignant melanoma cells. Once bound, it can mediate destruction of these cells through both complement-mediated lysis and antibody-dependent cellular cytotoxicity. Agents such as interleukin 2 (IL-2), which can augment effector cell function and promote destruction of antibody-coated tumor cells, might produce improved antitumor responses when combined with R24. In this series, the authors evaluated the combination of R24 and IL-2 in a Phase 1b study in patients with metastatic melanoma. Twenty-eight patients with metastatic melanoma were entered into the protocol at two institutions. Patients received 8 wk of IL-2 by continuous i.v. infusion at a dose (4.5 + 105 Amgen units/M2/day) designed to selectively expand natural killer (NK) cells. In weeks 5 and 6, patients received R24 for a total of four doses. Twenty-four h after each R24 infusion, patients received a 2-h bolus dose of IL-2 to help promote activity of NK effectors against antibody-coated melanoma targets. Addnl. IL-2 boluses were administered in weeks 7 and 8. Doses were escalated through two bolus doses of R24 (5 or 15 mg/M2) and two bolus doses of IL-2 (2.5 or 5.0 + 105 units/M2). Although one patient experienced severe capillary leak syndrome during IL-2, therapy was otherwise well tolerated. At the higher dose level of R24, two of four patients experienced transient but severe abdominal and chest discomfort, necessitating dose reduction One patient with ocular melanoma and liver metastases had a partial response. Two addnl. patients had minor responses. A dramatic increase in NK cell number was noted as a result of treatment, as was augmentation of cytolytic activity against cultured NK-sensitive targets. Antibody-dependent cellular cytotoxicity against cultured melanoma cells in the presence of exogenous R24 or in the presence of serum obtained from patients following R24 infusion also increased during treatment. The authors' experience indicates that R24 and low-dose IL-2 can be safely combined in patients with metastatic melanoma and that this combination can promote destruction of cultured melanoma cells. The clin. activity of this combination against ocular melanoma may merit further investigation.
AN 1997:72780 HCAPLUS <<LOGINID::20070726>>
DN 126:170199
TI Administration of R24 monoclonal antibody and low-dose interleukin 2 for malignant melanoma
AU Soiffer, Robert J.; Chapman, Paul B.; Murray, Christine; Williams, Linda; Unger, Paul; Collins, Heather; Houghton, Alan N.; Ritz, Jerome
CS Div. Hematological Malignancies, Dana-Farber Cancer Inst., Boston, MA,

02115, USA
SO Clinical Cancer Research (1997), 3(1), 17-24
CODEN: CCREF4; ISSN: 1078-0432
PB American Association for Cancer Research
DT Journal
LA English
RE.CNT 43 THERE ARE 43 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L31 ANSWER 9 OF 34 HCAPLUS COPYRIGHT 2007 ACS on STN
TI Levels of cell membrane CD59 regulate the extent of complement -mediated lysis of human melanoma cells
AB Normal and neoplastic cells are protected from autologous complement (C) attack by different cell-surface C-regulatory proteins including CD59 (protectin), CD46 (membrane cofactor protein) and CD55 (decay-accelerating factor). Indirect immunofluorescence (IIF) anal. showed a differential expression of CD59, CD46, and CD55 in nine human melanoma cell lines and that the expression of CD59 was highly heterogeneous compared with that of CD46 and CD55. Levels of cell membrane CD59 were found to regulate the differential sensitivity of melanoma cells investigated to homologous C-mediated lysis; in fact, an inverse correlation ($r > 0.7$) was found between levels of cell membrane CD59, but not of CD46 and CD55, and extent of C-mediated lysis of melanoma cells sensitized with scalar concns. of the anti-GD3 ganglioside mAb R24. Masking of CD59 by 2.5 μg/mL of the anti-CD59 mAb YTH53.1 induced or enhanced C-mediated lysis of melanoma cells sensitized with 2.5 μg/mL of mAb R24; the latter phenomenon was directly correlated ($r = 0.865$) with levels of cell membrane CD59. CD59 is bound to melanoma cells by a glycosylphosphatidylinositol anchor: treatment of C-resistant melanoma cells Mel 97, by increasing doses of phosphatidylinositol-specific phospholipase C (PI-PLC), progressively decreased cell-surface expression of CD59 and increased C-mediated lysis of cells sensitized with mAb R24. Staining of 38 benign and malignant lesions of melanocytic origin by mAb YTH53.1 demonstrated that CD59 is consistently expressed in vivo and confirmed the heterogeneous expression detected in vitro. The authors' data, altogether, demonstrate that CD59 is the main restriction factor of C-mediated lysis of melanoma cells and that levels of CD59 may account for their differential resistance to C-mediated lysis. The anal. of the levels of CD59 could represent an useful strategy in selecting melanoma patients who may benefit from immunotherapeutic treatment(s) that trigger C activation.
AN 1996:228193 HCAPLUS <<LOGINID::20070726>>
DN 124:286561
TI Levels of cell membrane CD59 regulate the extent of complement -mediated lysis of human melanoma cells
AU Brasoveanu, Lorelei Irina; Altomonte, Maresa; Fonsatti, Ester; Colizzi, Francesca; Coral, Sandra; Nicotra, Maria Rita; Cattarossi, Ilaria; Cattelan, Alessandro; Natali, Pier Giorgio; Maio, Michele
CS Advanced Immunotherapy Unit, Centro di Riferimento Oncologico, Aviano, 33081, Italy
SO Laboratory Investigation (1996), 74(1), 33-42
CODEN: LAINAW; ISSN: 0023-6837
PB Williams & Wilkins
DT Journal
LA English

L31 ANSWER 11 OF 34 HCAPLUS COPYRIGHT 2007 ACS on STN
TI Mapping effector functions of a monoclonal antibody to GD3 by characterization of a mouse-human chimeric antibody
AB R24, a mouse monoclonal antibody against GD3 ganglioside, exhibits a wide range of in vitro effector functions. It also has the ability to bind to itself, presumably through homophilic Fab-Fab interactions, which have been proposed to contribute to its high relative avidity for GD3 and to its effector function activity.

It is not known which of these characteristics is necessary for the antitumor effects observed in melanoma patients treated with R24. A mouse-human chimeric R24 (chR24) mol. has been constructed in which the GD3-binding site is preserved. Chimeric R24 demonstrates a lower level of binding to GD3 than does mouse R24, suggesting that there may be some differences between the GD3-binding sites of the two mAb or that Fc determinants can contribute to R24 avidity for GD3. The property of homophilic binding is retained by chR24, demonstrating formally that homophilic binding of R24 involves interactions between variable domains. Both R24 and chR24 fix human complement and mediate antibody-dependent cellular cytotoxicity, although chR24 was slightly less efficient at the latter. Unlike R24, chR24 was not able to inhibit melanoma cell attachment to plastic surfaces and was not able to activate human T lymphocytes. We hypothesize that chR24 does not bind to GD3 with an avidity high enough to mediate these effector functions.

AN 1995:235704 HCAPLUS <<LOGINID::20070726>>
DN 122:7515
TI Mapping effector functions of a monoclonal antibody to GD3 by characterization of a mouse-human chimeric antibody
AU Chapman, Paul B.; Gillies, Stephen D.; Houghton, Alan N.; Reilly, Regina M.
CS Memorial Sloan-Kettering Cancer Center, New York, NY, 10021, USA
SO Cancer Immunology Immunotherapy (1994), 39(3), 198-204
CODEN: CIIMDN; ISSN: 0340-7004
DT Journal
LA English

L31 ANSWER 17 OF 34 HCAPLUS COPYRIGHT 2007 ACS on STN
TI A mouse/human chimeric anti-(ganglioside GD3) antibody with enhanced antitumor activities
AB Ganglioside GD3, which is one of the major gangliosides expressed on the cell surface of human tumors of neuroectodermal origin has been focused on as a target mol. for passive immunotherapy. The authors have cloned the cDNA encoding the Ig light and heavy chains of an anti-GD3 monoclonal antibody KM641 (murine IgG3, κ), and constructed the chimeric genes by linking the cDNA fragments of the murine light and heavy variable regions to cDNA fragments of the human κ and γ1 constant regions, resp. The transfer of these cDNA constructs into SP2/0 mouse myeloma cells resulted in the production of the chimeric antibody, designated KM871, that retained specific binding activity to GD3. Indirect immunofluorescence revealed the same staining pattern for chimeric KM871 and the mouse counterpart KM641 on CD3-expressing melanoma cells. When human serum and human peripheral blood mononuclear cells were used as effectors in complement-mediated cytotoxicity and antibody-dependent cell-mediated cytotoxicity resp., the chimeric KM871 was more effective in killing GD3-expressing tumor cells than was the mouse counterpart KM641. The i.v. injection of chimeric KM871 markedly suppressed tumor growth in nude mice. The chimeric KM871, having enhanced antitumor activities and less immunogenicity than the mouse counterpart, would be a useful agent for passive immunotherapy of human cancer.
AN 1993:601424 HCAPLUS <<LOGINID::20070726>>
DN 119:201424
TI A mouse/human chimeric anti-(ganglioside GD3) antibody with enhanced antitumor activities
AU Shitara, Kenya; Kuwana, Yoshihisa; Nakamura, Kazuyasu; Tokutake, Yuko; Ohta, So; Miyaji, Hiromasa; Hasegawa, Mamoru; Hanai, Nobuo
CS Tokyo Res. Lab., Kyowa Hakko Kogyo Co. Ltd., Machida, 194, Japan
SO Cancer Immunology Immunotherapy (1993), 36(6), 373-80
CODEN: CIIMDN; ISSN: 0340-7004
DT Journal
LA English

L31 ANSWER 18 OF 34 HCAPLUS COPYRIGHT 2007 ACS on STN
TI Antitumor effects of a novel monoclonal antibody with high binding affinity to ganglioside GD3
AB Ganglioside GD3, which is one of the major gangliosides expressed on the cell surface of human tumors of neuroectodermal origin, was studied as a target mol. for passive immunotherapy. Ten anti-GD3 monoclonal antibodies (mAb) of the mouse IgG3 subclass were established by immunization with purified GD3 and melanoma cells. One of the established mAb, KM641, showed major reactivity with GD3 and minor reactivity with GQ1b out of 11 common gangliosides in an ELISA. Immunostaining of gangliosides, separated on TLC plates, using KM641 revealed that most of the melanoma cell lines contained immunoreactive GD3 and GD3-lactone at a high level, but only the adrenal gland and the urinary bladder out of 21 human normal tissues had immunoreactive GD3. In immunofluorescence, KM641 bound to a variety of live tumor cell lines especially melanoma cells, including some cell lines to which another anti-GD3 mAb R24, established previously, failed to bind. High-affinity binding of KM641 to a tumor cell line was quantified by Scatchard anal. (Kd = 1.9+10-8 M). KM641 exerted tumor-killing activity in the presence of effector cells or complement against melanoma cells expressing GD3 at a high level. Not only natural killer cells but also polymorphonuclear cells were effective as the effector cells in antibody-dependent cellular cytotoxicity. The i.v. injection of KM641 markedly suppressed the tumor growth of a slightly pos. cell line, C24.22 (7.2+105 binding sites/cell), as well as a very GD3-pos. cell line, G361 (1.9+107 binding sites/cell), inoculated intradermally in nude mice. KM641, characterized by a high binding affinity for GD3, has the potential to be a useful agent for passive immunotherapy of human cancer.
AN 1993:253101 HCAPLUS <<LOGINID::20070726>>
DN 118:253101
TI Antitumor effects of a novel monoclonal antibody with high binding affinity to ganglioside GD3
AU Ohta, So; Honda, Ayumi; Tokutake, Yuko; Yoshida, Hajime; Hanai, Nobuo
CS Tokyo Res. Lab., Kyowa Hakko Kogyo Co. Ltd., Machida, 194, Japan
SO Cancer Immunology Immunotherapy (1993), 36(4), 260-6
CODEN: CIIMDN; ISSN: 0340-7004
DT Journal
LA English

L31 ANSWER 21 OF 34 HCAPLUS COPYRIGHT 2007 ACS on STN
TI Immunorecognition of ganglioside epitopes: correlation between affinity and cytotoxicity of ganglioside antibodies
AB Cell-surface gangliosides have immunomodulatory effects that are presumed to play a role in tumor growth, progression, metastasis, and therapy. To study the epitopes of gangliosides on human malignant melanomas and to search for monoclonal antibodies (Mabs) with superior immunol. effector functions, 19 ganglioside antibodies were established. Specificity and affinity of 9 antibodies of IgG3 isotype were evaluated by ELISA and thin layer chromatog. with a panel of purified gangliosides. All antibodies recognized the ganglioside GD3, but their epitope specificity divided them into 5 groups. Their affinity consts. for ganglioside GD3 ranged from 4.7 + 106 to 2.3 + 108, with 2 + 107 for Mab R-24. Two antibodies possessed a higher affinity for GD2 than for GD3. The functional properties of the antibodies were investigated in vitro. Differences in the degree of tumor lysis by complement fixation correlated with the affinity consts. Every ganglioside antibody differed in epitope recognition, affinity, and cytotoxicity. Therefore some of these antibodies might even be more useful in the immunotherapy of malignant melanoma than Mab R-24.
AN 1992:610375 HCAPLUS <<LOGINID::20070726>>
DN 117:210375
TI Immunorecognition of ganglioside epitopes: correlation between affinity

and cytotoxicity of ganglioside antibodies
AU Dippold, Wolfgang; Bernhard, Helga
CS Med. Klin., Johannes Gutenberg-Univ., Mainz, D-6500, Germany
SO Eur. J. Cancer, Part A (1992), 28A(10), 1605-10
CODEN: EJCTEA
DT Journal
LA English

=> d l32 1-2 ti abs bib
YOU HAVE REQUESTED DATA FROM FILE 'HCAPLUS' - CONTINUE? (Y)/N:y

L32 ANSWER 1 OF 2 HCAPLUS COPYRIGHT 2007 ACS on STN
TI Mechanisms of anti-lung cancer activity for monoclonal antibody to epidermal growth factor receptor
AB The objective of this study was to examine the mechanisms of anti-lung cancer activity for monoclonal antibody to epidermal growth factor receptor (EGFR). Cytotoxicity was observed by MTT assay and expression of EGFR on the surface of xenografts cells was investigated using immunohistochem. method. Complement -dependent cytotoxicity (CDC) of EGFR monoclonal antibody egf/r3(IgG2a) to lung cancer cells was observed, whereas no antibody-dependent LAK cell-mediated cytotoxicity (ADCC) was found, which may be due to isotype of egf/r3 antibody. The expression level of EGFR on xenografts lung tumor cell in nude mice was down regulated on day 4 after egf/r3 McAb immunotherapy and returned to original level on day 15 after administration of egf/r3. Possible mechanisms for egf/r3 McAb mediated antitumor activity are: (1) down regulation of EGFR expression on tumor cell via internalization of complex of the egf/r3 McAb with EGFR, (2) blocking the binding of EGF to EGFR and inhibiting protein tyrosine kinase activity of the receptor, and (3) CDC mediated antitumor activity.
AN 1998:264458 HCAPLUS <<LOGINID::20070726>>
DN 129:53210
TI Mechanisms of anti-lung cancer activity for monoclonal antibody to epidermal growth factor receptor
AU Ren, Xinling; Jin, Boquan; Shen, Liyin; Ma, Jin
CS Xijing Hosp., Fourth Military Medical Univ., Xi'an, 710033, Peop. Rep. China
SO Disi Junyi Daxue Xuebao (1997), 18(6), 560-562
CODEN: DJDXEG; ISSN: 1000-2790
PB Disi Junyi Daxue Xuebao Bianjibu
DT Journal
LA Chinese

L32 ANSWER 2 OF 2 HCAPLUS COPYRIGHT 2007 ACS on STN
TI Modification of monoclonal antibody carbohydrates by oxidation, conjugation, or deoxymannojirimycin does not interfere with antibody effector functions
AB Site-specific attachment of metal chelators or cytotoxic agents to the carbohydrate region of monoclonal antibodies results in clin. useful immunoconjugates [Doerr et al. (1991), Wynant et al. (1991)]. Since the capacity of monoclonal antibodies (mAb) to mediate tumor cell lysis via antibody-dependent cellular cytotoxicity (ADCC) or complement -dependent cytotoxicity (CDC) may accentuate the therapeutic effectiveness of immunoconjugates, the authors determined whether site-specific modification of mAb carbohydrates interfered with these functions. The chemical modifications examined consisted of periodate oxidation and subsequent conjugation to either a peptide linker/chelator (GYK-DTPA) or a cytotoxic drug (doxorubicin adipic dihydrazide). MAb-associated carbohydrates were also modified metabolically by incubating hybridoma cells in the presence of a glucosidase inhibitor deoxymannojirimycin to produce high-mannose

antibody. All four forms (unaltered, oxidized, conjugated and high-mannose) of murine mAb OVB-3 mediated tumor cell lysis via CDC. Similarly, equivalent ADCC was observed with native and conjugated forms of mAb OVB-3 and EGFR.1. ADCC was achieved with different murine effector cells such as naive (NS), poly (I:C)- and lipopolysaccharide-stimulated (SS) spleen cells, or Corynebacterium-parvum-elicited peritoneal cells (PEC). All murine effector cell types mediated tumor cell lysis but differed in potency such that PEC>SS>NS. Excellent ADCC activity was also demonstrable by human peripheral blood mononuclear cells with OVB-3-GYK-DTPA and high-mannose OVB-3 mAb. ADCC activity was detectable in vivo: both native and conjugated OVB-3 inhibited growth of OVCAR-3 xenografts in nude mice primed with C. parvum. In conclusion, modification of mAb carbohydrates did not compromise their in vivo or in vitro biol. functions. Therefore, combination therapy using immunomodulators to enhance the effector functions of site-specific immunoconjugates could be seriously contemplated.

AN 1994:455506 HCAPLUS <<LOGINID::20070726>>
DN 121:55506
TI Modification of monoclonal antibody carbohydrates by oxidation, conjugation, or deoxymannojirimycin does not interfere with antibody effector functions
AU Awwad, Michel; Strome, Phoebe G.; Gilman, Steven C.; Axelrod, Helena R.
CS Dep. Biol. Res., CYTOGEN Corp., Princeton, NJ, 08540, USA
SO Cancer Immunology Immunotherapy (1994), 38(1), 23-30
CODEN: CIIMDN; ISSN: 0340-7004
DT Journal
LA English

=> d l27 1-3 ti abs bib
YOU HAVE REQUESTED DATA FROM FILE 'HCAPLUS' - CONTINUE? (Y)/N:y

L27 ANSWER 1 OF 3 HCAPLUS COPYRIGHT 2007 ACS on STN
TI Methods and compositions for the prevention or treatment of neoplasia comprising a COX-2 inhibitor in combination with an epidermal growth factor receptor antagonist
AB The present invention relates to a novel method of preventing and/or treating neoplasia disorders in a subject that is in need of such prevention or treatment by administering to the subject at least one COX-2 inhibitor in combination with an EGF receptor antagonist. Compns., pharmaceutical compns. and kits are also described.
AN 2004:533970 HCAPLUS <<LOGINID::20070726>>
DN 141:65088
TI Methods and compositions for the prevention or treatment of neoplasia comprising a COX-2 inhibitor in combination with an epidermal growth factor receptor antagonist
IN Masferrer, Jaime
PA Pharmacia Corporation, USA
SO U.S. Pat. Appl. Publ., 103 pp., Cont.-in-part of U.S. Ser. No. 470,951.
CODEN: USXXCO
DT Patent
LA English
FAN.CNT 21

	PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI	US 2004127470	A1	20040701	US 2003-651916	20030829 <--
	EP 1522313	A1	20050413	EP 2004-26577	19991222 <--
	R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, FI, RO, CY				
	WO 2005037259	A2	20050428	WO 2004-US27574	20040825
	WO 2005037259	A3	20050804		
	W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BW, BY, BZ, CA, CH,				

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GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC,
LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NA, NI,
NO, NZ, OM, PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SY,
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RW: BW, GH, GM, KE, LS, MW, MZ, NA, SD, SL, SZ, TZ, UG, ZM, ZW, AM,
AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK,
EE, ES, FI, FR, GB, GR, HU, IE, IT, LU, MC, NL, PL, PT, RO, SE,
SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE,
SN, TD, TG

	AU 2004210578	A1	20041007	AU 2004-210578	20040910 <--
PRAI	US 1998-113786P	P	19981223	<--	
	US 1999-470951	B2	19991222	<--	
	US 1999-385214	A	19990827	<--	
	AU 2000-25936	A3	19991222	<--	
	EP 1999-968939	A3	19991222	<--	
	US 2003-651916	A	20030829		

L27 ANSWER 2 OF 3 HCAPLUS COPYRIGHT 2007 ACS on STN
TI Method for relieving pain associated with an internal disease site
AB Methods are provided for in vivo administration of a pain-relieving drug, such as a local anesthetic (e.g. lidocaine), to an interior disease site for pain relief at the interior disease site. In the invention pain treatment methods, a subject is administered a targeting construct comprising a biol. compatible pain-relieving agent and a tumor-avid ligand or monoclonal antibody that preponderantly binds to or is taken up by the tissue associated with an interior disease site. Administration is by a method other than topical injection or application, such as parenteral injection. Because the pain-relieving agent is delivered by the ligand to the disease site, intractable pain situated in the interior of the body, such as is caused by various tumors, can be managed using a lower level of the pain-relieving agent then is required when the pain-relieving agent is injected in the free state.
AN 2001:489224 HCAPLUS <<LOGINID::20070726>>
DN 135:97445
TI Method for relieving pain associated with an internal disease site
IN Luiken, George A.
PA Fluoro Probe, Inc., USA
SO PCT Int. Appl., 31 pp.
CODEN: PIXXD2
DT Patent
LA English
FAN.CNT 1

	PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI	WO 2001047512	A2	20010705	WO 2000-US42661	20001206 <--
	WO 2001047512	A3	20020502		

W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN,
CR, CU, CZ, DE, DK, DM, DZ, EE, ES, FI, GB, GD, GE, GH, GM, HR,
HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT,
LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, PL, PT, RO, RU,
SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, US, UZ, VN,
YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY,
DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR, BF,
BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG

	AU 2001049041	A5	20010709	AU 2001-49041	20001206 <--
PRAI	US 1999-457498	A1	19991208	<--	
	WO 2000-US42661	W	20001206		

L27 ANSWER 3 OF 3 HCAPLUS COPYRIGHT 2007 ACS on STN
TI Compositions and methods for improved detection and classification of neoplasms using antibodies to transcription factors
AB A first aspect of the present invention is composition including at least two

different specific binding members such as antibodies or active fragments thereof that specifically bind with at least two different transcription factors. A second aspect of the present invention is a method of diagnosing a neoplasm or malignancy or prognosing the course of a malignancy or treatment thereof including contacting at least one specific binding member that specifically binds with a transcription factor with a sample, and detecting the binding of said specific binding member with a transcription factor in said sample. A third aspect of the present invention is a method for identifying a test compound that modulates a neoplasm or malignancy including contacting a sample with at least one test compound, contacting said sample with at least one specific binding member that binds with at least one transcription factor, and detecting the binding of said at least one specific binding member with at least one transcription factor. Twenty-one paraffin-embedded tumors were stained with anti-MyoD monoclonal antibody 12. Seven of seven rhabdomyosarcoma detectably stained with that monoclonal antibody while fourteen of fourteen non-rhabdomyosarcoma tumors, including non-Hodgkin's lymphomas, neuroblastomas and Ewings sarcomas did not detectably stain with that monoclonal antibody.

AN 2001:228673 HCAPLUS <<LOGINID::20070726>>
DN 134:249225
TI Compositions and methods for improved detection and classification of neoplasms using antibodies to transcription factors
IN Dias, Peter; Singh, Sujay
PA Imgenex Corporation, USA
SO PCT Int. Appl., 70 pp.
CODEN: PIXXD2
DT Patent
LA English
FAN.CNT 1

	PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI	WO 2001021136	A2	20010329	WO 2000-US26105	20000923 <--
	WO 2001021136	A3	20011004		
	W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CR, CU, CZ, DE, DK, DM, DZ, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM				
	RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG				
	US 6623936	B1	20030923	US 2000-499559	20000207 <--
	AU 2001011899	A	20010424	AU 2001-11889	20000923 <--
PRAI	US 1999-155972P	P	19990924 <--		
	US 2000-499559	A	20000207		
	WO 2000-US26105	W	20000923		

=> d l27 1-5 ti abs bib
YOU HAVE REQUESTED DATA FROM FILE 'HCAPLUS' - CONTINUE? (Y)/N:y

L27 ANSWER 1 OF 3 HCAPLUS COPYRIGHT 2007 ACS on STN
TI Methods and compositions for the prevention or treatment of neoplasia comprising a COX-2 inhibitor in combination with an epidermal growth factor receptor antagonist
AB The present invention relates to a novel method of preventing and/or treating neoplasia disorders in a subject that is in need of such prevention or treatment by administering to the subject at least one COX-2 inhibitor in combination with an EGF receptor antagonist. Compns.,

pharmaceutical compns. and kits are also described.
AN 2004:533970 HCAPLUS <<LOGINID::20070726>>
DN 141:65088
TI Methods and compositions for the prevention or treatment of neoplasia comprising a COX-2 inhibitor in combination with an epidermal growth factor receptor antagonist
IN Masferrer, Jaime
PA Pharmacia Corporation, USA
SO U.S. Pat. Appl. Publ., 103 pp., Cont.-in-part of U.S. Ser. No. 470,951.
CODEN: USXXCO
DT Patent
LA English
FAN.CNT 21

	PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI	US 2004127470	A1	20040701	US 2003-651916	20030829 <--
	EP 1522313	A1	20050413	EP 2004-26577	19991222 <--
	R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, FI, RO, CY				
	WO 2005037259	A2	20050428	WO 2004-US27574	20040825
	WO 2005037259	A3	20050804		
	W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BW, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NA, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW				
	RW: BW, GH, GM, KE, LS, MW, MZ, NA, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IT, LU, MC, NL, PL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG				
	AU 2004210578	A1	20041007	AU 2004-210578	20040910 <--
PRAI	US 1998-113786P	P	19981223	<--	
	US 1999-470951	B2	19991222	<--	
	US 1999-385214	A	19990827	<--	
	AU 2000-25936	A3	19991222	<--	
	EP 1999-968939	A3	19991222	<--	
	US 2003-651916	A	20030829		

L27 ANSWER 2 OF 3 HCAPLUS COPYRIGHT 2007 ACS on STN
TI Method for relieving pain associated with an internal disease site
AB Methods are provided for in vivo administration of a pain-relieving drug, such as a local anesthetic (e.g. lidocaine), to an interior disease site for pain relief at the interior disease site. In the invention pain treatment methods, a subject is administered a targeting construct comprising a biol. compatible pain-relieving agent and a tumor-avid ligand or monoclonal antibody that preponderantly binds to or is taken up by the tissue associated with an interior disease site. Administration is by a method other than topical injection or application, such as parenteral injection. Because the pain-relieving agent is delivered by the ligand to the disease site, intractable pain situated in the interior of the body, such as is caused by various tumors, can be managed using a lower level of the pain-relieving agent then is required when the pain-relieving agent is injected in the free state.
AN 2001:489224 HCAPLUS <<LOGINID::20070726>>
DN 135:97445
TI Method for relieving pain associated with an internal disease site
IN Luiken, George A.
PA Fluoro Probe, Inc., USA
SO PCT Int. Appl., 31 pp.
CODEN: PIXXD2
DT Patent
LA English

FAN.CNT 1

	PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI	WO 2001047512	A2	20010705	WO 2000-US42661	20001206 <--
	WO 2001047512	A3	20020502		

W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CR, CU, CZ, DE, DK, DM, DZ, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM

RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG

	AU 2001049041	A5	20010709	AU 2001-49041	20001206 <--
PRAI	US 1999-457498	A1	19991208	<--	
	WO 2000-US42661	W	20001206		

L27 ANSWER 3 OF 3 HCAPLUS COPYRIGHT 2007 ACS on STN

TI Compositions and methods for improved detection and classification of neoplasms using antibodies to transcription factors

AB A first aspect of the present invention is composition including at least two different specific binding members such as antibodies or active fragments thereof that specifically bind with at least two different transcription factors. A second aspect of the present invention is a method of diagnosing a neoplasm or malignancy or prognosing the course of a malignancy or treatment thereof including contacting at least one specific binding member that specifically binds with a transcription factor with a sample, and detecting the binding of said specific binding member with a transcription factor in said sample. A third aspect of the present invention is a method for identifying a test compound that modulates a neoplasm or malignancy including contacting a sample with at least one test compound, contacting said sample with at least one specific binding member that binds with at least one transcription factor, and detecting the binding of said at least one specific binding member with at least one transcription factor. Twenty-one paraffin-embedded tumors were stained with anti-MyoD monoclonal antibody 12. Seven of seven rhabdomyosarcoma detectably stained with that monoclonal antibody while fourteen of fourteen non-rhabdomyosarcoma tumors, including non-Hodgkin's lymphomas, neuroblastomas and Ewings sarcomas did not detectably stain with that monoclonal antibody.

AN 2001:228673 HCAPLUS <<LOGINID::20070726>>

DN 134:249225

TI Compositions and methods for improved detection and classification of neoplasms using antibodies to transcription factors

IN Dias, Peter; Singh, Sujay

PA Imgenex Corporation, USA

SO PCT Int. Appl., 70 pp.
CODEN: PIXXD2

DT Patent

LA English

FAN.CNT 1

	PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI	WO 2001021136	A2	20010329	WO 2000-US26105	20000923 <--
	WO 2001021136	A3	20011004		

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RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ,

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                                                        ENTRY    SESSION
CA SUBSCRIBER PRICE                                      0.00     -19.50

 SESSION WILL BE HELD FOR 120 MINUTES
STN INTERNATIONAL SESSION SUSPENDED AT 10:09:24 ON 26 JUL 2007

Connecting via Winsock to STN



Welcome to STN International!  Enter x:x

LOGINID:SSPTAEXO1623

PASSWORD:
 * * * * * * RECONNECTED TO STN INTERNATIONAL * * * * * *
SESSION RESUMED IN FILE 'STNGUIDE' AT 10:10:45 ON 26 JUL 2007
FILE 'STNGUIDE' ENTERED AT 10:10:45 ON 26 JUL 2007
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COST IN U.S. DOLLARS                                SINCE FILE      TOTAL
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FULL ESTIMATED COST                                       0.06     146.94

DISCOUNT AMOUNTS (FOR QUALIFYING ACCOUNTS)          SINCE FILE      TOTAL
                                                         ENTRY    SESSION
CA SUBSCRIBER PRICE                                       0.00     -19.50

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YOU HAVE REQUESTED DATA FROM FILE 'HCAPLUS' - CONTINUE? (Y)/N:y


L28  ANSWER 1 OF 5  HCAPLUS  COPYRIGHT 2007 ACS on STN
TI   Methods and compositions for the prevention or treatment of neoplasia
     comprising a COX-2 inhibitor in combination with an epidermal growth
     factor receptor antagonist

L28  ANSWER 2 OF 5  HCAPLUS  COPYRIGHT 2007 ACS on STN
TI   Method for relieving pain associated with an internal disease site

L28  ANSWER 3 OF 5  HCAPLUS  COPYRIGHT 2007 ACS on STN
TI   Process for detecting, extracting or removing human or mammalian cells
     with a disturbed cellular cycle regulation or unlimited proliferation or
     tumor-forming ability

L28  ANSWER 4 OF 5  HCAPLUS  COPYRIGHT 2007 ACS on STN
TI   Receptor protein and its use

L28  ANSWER 5 OF 5  HCAPLUS  COPYRIGHT 2007 ACS on STN
TI   Analysis of proliferative grade using anti-PCNA/cyclin monoclonal
     antibodies in fixed, embedded tissues.  Comparison with flow cytometric
     analysis


=> d l28 1-5 ti 3 4 5 ti abs bib
YOU HAVE REQUESTED DATA FROM FILE 'HCAPLUS' - CONTINUE? (Y)/N:y


L28  ANSWER 1 OF 5  HCAPLUS  COPYRIGHT 2007 ACS on STN
TI   Methods and compositions for the prevention or treatment of neoplasia
     comprising a COX-2 inhibitor in combination with an epidermal growth
```

factor receptor antagonist

TI Methods and compositions for the prevention or treatment of neoplasia comprising a COX-2 inhibitor in combination with an epidermal growth factor receptor antagonist

AB The present invention relates to a novel method of preventing and/or treating neoplasia disorders in a subject that is in need of such prevention or treatment by administering to the subject at least one COX-2 inhibitor in combination with an EGF receptor antagonist. Compns., pharmaceutical compns. and kits are also described.

AN 2004:533970 HCAPLUS <<LOGINID::20070726>>

DN 141:65088

TI Methods and compositions for the prevention or treatment of neoplasia comprising a COX-2 inhibitor in combination with an epidermal growth factor receptor antagonist

IN Masferrer, Jaime

PA Pharmacia Corporation, USA

SO U.S. Pat. Appl. Publ., 103 pp., Cont.-in-part of U.S. Ser. No. 470,951.
CODEN: USXXCO

DT Patent

LA English

FAN.CNT 21

	PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI	US 2004127470	A1	20040701	US 2003-651916	20030829 <--
	EP 1522313	A1	20050413	EP 2004-26577	19991222 <--
	R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, FI, RO, CY				
	WO 2005037259	A2	20050428	WO 2004-US27574	20040825
	WO 2005037259	A3	20050804		
	W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BW, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NA, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW				
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	AU 2004210578	A1	20041007	AU 2004-210578	20040910 <--
PRAI	US 1998-113786P	P	19981223 <--		
	US 1999-470951	B2	19991222 <--		
	US 1999-385214	A	19990827 <--		
	AU 2000-25936	A3	19991222 <--		
	EP 1999-968939	A3	19991222 <--		
	US 2003-651916	A	20030829		

L28 ANSWER 2 OF 5 HCAPLUS COPYRIGHT 2007 ACS on STN

TI Method for relieving pain associated with an internal disease site

TI Method for relieving pain associated with an internal disease site

AB Methods are provided for in vivo administration of a pain-relieving drug, such as a local anesthetic (e.g. lidocaine), to an interior disease site for pain relief at the interior disease site. In the invention pain treatment methods, a subject is administered a targeting construct comprising a biol. compatible pain-relieving agent and a tumor-avid ligand or monoclonal antibody that preponderantly binds to or is taken up by the tissue associated with an interior disease site. Administration is by a method other than topical injection or application, such as parenteral injection. Because the pain-relieving agent is delivered by the ligand to the disease site, intractable pain situated in the interior of the body, such as is caused by various tumors, can be managed using a lower level of the pain-relieving agent then is required when the pain-relieving agent is injected in the free state.

AN 2001:489224 HCAPLUS <<LOGINID::20070726>>
DN 135:97445
TI Method for relieving pain associated with an internal disease site
IN Luiken, George A.
PA Fluoro Probe, Inc., USA
SO PCT Int. Appl., 31 pp.
CODEN: PIXXD2
DT Patent
LA English
FAN.CNT 1

	PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI	WO 2001047512	A2	20010705	WO 2000-US42661	20001206 <--
	WO 2001047512	A3	20020502		
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	RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG				
	AU 2001049041	A5	20010709	AU 2001-49041	20001206 <--
PRAI	US 1999-457498	A1	19991208	<--	
	WO 2000-US42661	W	20001206		

L28 ANSWER 3 OF 5 HCAPLUS COPYRIGHT 2007 ACS on STN
TI Process for detecting, extracting or removing human or mammalian cells with a disturbed cellular cycle regulation or unlimited proliferation or tumor-forming ability
TI Process for detecting, extracting or removing human or mammalian cells with a disturbed cellular cycle regulation or unlimited proliferation or tumor-forming ability
AB For detecting, identifying, extracting or removing human or animal cells with a disturbed cellular cycle regulation or unlimited proliferation or tumor-forming ability, the presence of an association of cdc37 protein with extrachromosomal nucleic acid is detected in cells or tissue fluids. This can be done, for example, by using a detectable substance which can specifically bind to the associate, a nucleic acid or oligonucleotide which hybridizes with the nucleic acid of the association or binding substances immobilized on a solid substrate. This latter method also makes it possible to extract or remove such cells. Thus the "heteromer" cdc37 protein-DNA complex from MCF-7 mammalian carcinoma cells was isolated, cloned and expressed in E.coli, the DNA was sequenced. Similarly protein-DNA complexes were isolated from colon cancer, Hodgkin-lymphoma, melanoma and acute myeloid leukemia cells; sequences that may be associated with these are reported. Mice were boosted with the cdc protein-DNA complex isolated from MCF-7; after 62 days, the spleen lymphocytes were isolated and used for the production of hybridoma cells; after repeated selection and subcloning the hybridoma clone 3D6 monoclonal antibody was obtained. The monoclonal antibody 3D6 specific to the tumor cdc37-DNA complex was used to identify tumor cells in cell lysate, in tumor biopsies, on the surface of MCF-7 carcinoma cells and in the serum of tumor patients. Tumor cells were concentrated from peripheral blood lymphocytes using the monoclonal antibodies and labeled secondary antibodies in conjunction with magnetic beads and FACS technique. Tumors cells can be separated from the blood of malignant melanoma patients using immobilized antibodies on a Sepharose column. The cdc37-DNA complex can be detected by in situ hybridization or PCR. The invention also includes peptides that inhibit the in vivo formation of the cdc37-DNA complex; the application of the complex and the monoclonal antibody

for pharmaceutical usage.
AN 1999:249109 HCAPLUS <<LOGINID::20070726>>
DN 130:293622
TI Process for detecting, extracting or removing human or mammalian cells with a disturbed cellular cycle regulation or unlimited proliferation or tumor-forming ability
IN Abken, Hinrich
PA Germany
SO PCT Int. Appl., 106 pp.
CODEN: PIXXD2
DT Patent
LA German
FAN.CNT 1

	PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI	WO 9918235	A1	19990415	WO 1998-EP6384	19981007 <--
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	RW: AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE				
	DE 19821506	A1	19990415	DE 1998-19821506	19980513 <--
	EP 1021564	A1	20000726	EP 1998-954373	19981007 <--
	R: AT, CH, DE, DK, ES, FR, GB, IT, LI				
	JP 2001519169	T	20011023	JP 2000-515027	19981007 <--
PRAI	DE 1997-19744335	A	19971007 <--		
	DE 1997-19749118	A	19971106 <--		
	DE 1998-19821506	A	19980513 <--		
	WO 1998-EP6384	W	19981007 <--		

RE.CNT 8 THERE ARE 8 CITED REFERENCES AVAILABLE FOR THIS RECORD
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L28 ANSWER 4 OF 5 HCAPLUS COPYRIGHT 2007 ACS on STN
TI Receptor protein and its use
TI Receptor protein and its use
AB A receptor protein derived from human dendritic cell, its partial peptide and their salts are disclosed. The dendritic cell receptor protein belongs to TNF receptor family. A production process of the receptor protein, an antibody against the receptor protein, a method for determination of a ligand to the receptor protein, a screening method and a screening kit for a compound which alters binding properties between a ligand and the receptor protein as well as a pharmaceutical composition of such compound are also disclosed. The receptor protein derived from human dendritic cell, its partial peptide and their salts are useful as reagents for screening ligands, agonists, antagonists and the like. The antibody is useful as a reagent for quant. anal. of the receptor protein in a specimen fluid. Compns. containing compound that alters binding of the receptor protein and its ligand are useful for preventing and treating cancer, AIDS, infections, allergic immunol. diseases, inflammation, autoimmune diseases, bronchial asthma, sepsis, tuberculosis, etc.
AN 1998:708734 HCAPLUS <<LOGINID::20070726>>
DN 129:329705
TI Receptor protein and its use
IN Nishi, Kazunori; Shintani, Atsushi; Horiguchi, Takashi
PA Takeda Chemical Industries, Ltd., Japan
SO Eur. Pat. Appl., 65 pp.
CODEN: EPXXDW
DT Patent
LA English
FAN.CNT 1

	PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI	EP 873998	A2	19981028	EP 1998-303190	19980424 <--
	EP 873998	A3	20000614		
	R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT,				

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L14             0 L8 AND L9 AND L10

=> s l11 and l12 and l13

L15            10 L11 AND L12 AND L13

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FILE COVERS 1907 - 26 Jul 2007  VOL 147 ISS 5
FILE LAST UPDATED: 25 Jul 2007  (20070725/ED)

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L16             2 L8 AND L9

=> s l8 and l10

L17             0 L8 AND L10

=> s l9 and l10

L18            57 L9 AND L10

=> file stnguide

COST IN U.S. DOLLARS                        SINCE FILE      TOTAL
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IE, SI, LT, LV, FI, RO

	CA 2229449	A1	19981025	CA 1998-2229449	19980423 <--
	JP 11152300	A	19990608	JP 1998-114450	19980424 <--
PRAI	JP 1997-109798	A	19970425 <--		
	JP 1997-251867	A	19970917 <--		

L28 ANSWER 5 OF 5 HCAPLUS COPYRIGHT 2007 ACS on STN
TI Analysis of proliferative grade using anti-PCNA/cyclin monoclonal antibodies in fixed, embedded tissues. Comparison with flow cytometric analysis
TI Analysis of proliferative grade using anti-PCNA/cyclin monoclonal antibodies in fixed, embedded tissues. Comparison with flow cytometric analysis
AB Monoclonal antibody 19A2, generated to PCNA/cyclin, a 36-kilodaltons, S-phase-associated nuclear protein, was used to identify proliferating cells within fixed, embedded tissue sections. Deparaffinized sections of 41 methacarn-fixed human tumors were immunostained with 19A2 by using a streptavidin biotin immunoperoxidase system. A semiquant. scoring system was used to evaluate the fraction of cells that were PCNA/cyclin-pos., and this score was compared with cell kinetic data obtained from parallel flow cytometric S-phase anal. that was performed on fresh samples of the same tumors. While there was general agreement between the slide-based, antibody-derived and the flow cytometrically derived cell kinetic information, some discrepancies were observed Some of the latter represented cases in which the anti-PCNA/cyclin antibody prepns. demonstrated significant heterogeneity in the nos. of proliferating cells in different regions of the tumor. In other cases, a significant fraction of the pos. cells corresponded to nontumor stromal and/or inflammatory cells. In these cases, the slide-based method provided more information about the tumor cell population than did the flow cytometry data. Semiquant. immunocytochem. anal. with anti-PCNA/cyclin antibodies may represent a simple, reproducible, yet powerful technique for the routine anal. of cell kinetic data in alc.-fixed, paraffin-embedded tissue.
AN 1989:530129 HCAPLUS <<LOGINID::20070726>>
DN 111:130129
TI Analysis of proliferative grade using anti-PCNA/cyclin monoclonal antibodies in fixed, embedded tissues. Comparison with flow cytometric analysis
AU Garcia, Rochelle L.; Coltrera, Marc D.; Gown, Allen M.
CS Dep. Pathol., Univ. Washington, Seattle, WA, USA
SO American Journal of Pathology (1989), 134(4), 733-9
CODEN: AJPAA4; ISSN: 0002-9440
DT Journal
LA English

L28 ANSWER 3 OF 5 HCAPLUS COPYRIGHT 2007 ACS on STN
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protein-DNA complexes were isolated from colon cancer, Hodgkin-lymphoma, melanoma and acute myeloid leukemia cells; sequences that may be associated with these are reported. Mice were boosted with the cdc protein-DNA complex isolated from MCF-7; after 62 days, the spleen lymphocytes were isolated and used for the production of hybridoma cells; after repeated selection and subcloning the hybridoma clone 3D6 monoclonal antibody was obtained. The monoclonal antibody 3D6 specific to the tumor cdc37-DNA complex was used to identify tumor cells in cell lysate, in tumor biopsies, on the surface of MCF-7 carcinoma cells and in the serum of tumor patients. Tumor cells were concentrated from peripheral blood lymphocytes using the monoclonal antibodies and labeled secondary antibodies in conjunction with magnetic beads and FACS technique. Tumors cells can be separated from the blood of malignant melanoma patients using immobilized antibodies on a Sepharose column. The cdc37-DNA complex can be detected by in situ hybridization or PCR. The invention also includes peptides that inhibit the in vivo formation of the cdc37-DNA complex; the application of the complex and the monoclonal antibody for pharmaceutical usage.

AN 1999:249109 HCAPLUS <<LOGINID::20070726>>
DN 130:293622
TI Process for detecting, extracting or removing human or mammalian cells with a disturbed cellular cycle regulation or unlimited proliferation or tumor-forming ability
IN Abken, Hinrich
PA Germany
SO PCT Int. Appl., 106 pp.
CODEN: PIXXD2
DT Patent
LA German
FAN.CNT 1

	PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI	WO 9918235	A1	19990415	WO 1998-EP6384	19981007 <--
	W: JP, US				
	RW: AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE				
	DE 19821506	A1	19990415	DE 1998-19821506	19980513 <--
	EP 1021564	A1	20000726	EP 1998-954373	19981007 <--
	R: AT, CH, DE, DK, ES, FR, GB, IT, LI				
	JP 2001519169	T	20011023	JP 2000-515027	19981007 <--
PRAI	DE 1997-19744335	A	19971007 <--		
	DE 1997-19749118	A	19971106 <--		
	DE 1998-19821506	A	19980513 <--		
	WO 1998-EP6384	W	19981007 <--		

RE.CNT 8 THERE ARE 8 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L28 ANSWER 4 OF 5 HCAPLUS COPYRIGHT 2007 ACS on STN
TI Receptor protein and its use
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AB A receptor protein derived from human dendritic cell, its partial peptide and their salts are disclosed. The dendritic cell receptor protein belongs to TNF receptor family. A production process of the receptor protein, an antibody against the receptor protein, a method for determination of a ligand to the receptor protein, a screening method and a screening kit for a compound which alters binding properties between a ligand and the receptor protein as well as a pharmaceutical composition of such compound are also disclosed. The receptor protein derived from human dendritic cell, its partial peptide and their salts are useful as reagents for screening ligands, agonists, antagonists and the like. The antibody is useful as a reagent for quant. anal. of the receptor protein in a specimen fluid.

Compns. containing compound that alters binding of the receptor protein and its ligand are useful for preventing and treating cancer, AIDS, infections, allergic immunol. diseases, inflammation, autoimmune diseases, bronchial asthma, sepsis, tuberculosis, etc.

AN 1998:708734 HCAPLUS <<LOGINID::20070726>>
DN 129:329705
TI Receptor protein and its use
IN Nishi, Kazunori; Shintani, Atsushi; Horiguchi, Takashi
PA Takeda Chemical Industries, Ltd., Japan
SO Eur. Pat. Appl., 65 pp.
CODEN: EPXXDW
DT Patent
LA English
FAN.CNT 1

	PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI	EP 873998	A2	19981028	EP 1998-303190	19980424 <--
	EP 873998	A3	20000614		
	R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO				
	CA 2229449	A1	19981025	CA 1998-2229449	19980423 <--
	JP 11152300	A	19990608	JP 1998-114450	19980424 <--
PRAI	JP 1997-109798	A	19970425 <--		
	JP 1997-251867	A	19970917 <--		

L28 ANSWER 5 OF 5 HCAPLUS COPYRIGHT 2007 ACS on STN
TI Analysis of proliferative grade using anti-PCNA/cyclin monoclonal antibodies in fixed, embedded tissues. Comparison with flow cytometric analysis
TI Analysis of proliferative grade using anti-PCNA/cyclin monoclonal antibodies in fixed, embedded tissues. Comparison with flow cytometric analysis
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DN 111:130129
TI Analysis of proliferative grade using anti-PCNA/cyclin monoclonal antibodies in fixed, embedded tissues. Comparison with flow cytometric analysis
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CS Dep. Pathol., Univ. Washington, Seattle, WA, USA
SO American Journal of Pathology (1989), 134(4), 733-9
CODEN: AJPAA4; ISSN: 0002-9440
DT Journal
LA English

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DISCOUNT AMOUNTS (FOR QUALIFYING ACCOUNTS)          SINCE FILE      TOTAL
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FILE LAST UPDATED: 25 Jul 2007  (20070725/ED)
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L33 36 L21 AND COMPLEMENT

=> s l23 and complement

69689 COMPLEMENT
L34 9 L23 AND COMPLEMENT

=> file stnguide

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FULL ESTIMATED COST	2.60	179.26

DISCOUNT AMOUNTS (FOR QUALIFYING ACCOUNTS)	SINCE FILE ENTRY	TOTAL SESSION
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=> d l33 1-36 ti
YOU HAVE REQUESTED DATA FROM FILE 'HCAPLUS' - CONTINUE? (Y)/N:y

L33 ANSWER 1 OF 36 HCAPLUS COPYRIGHT 2007 ACS on STN
TI The Ch14.18-GM-CSF fusion protein is effective at mediating antibody-dependent cellular cytotoxicity and complement -dependent cytotoxicity in vitro

L33 ANSWER 2 OF 36 HCAPLUS COPYRIGHT 2007 ACS on STN
TI Treatment of neoplastic meningeal xenografts by intraventricular administration of an anti-ganglioside monoclonal antibody, 3F8

L33 ANSWER 3 OF 36 HCAPLUS COPYRIGHT 2007 ACS on STN
TI Identification of a 220-kDa membrane tumor-associated antigen by human anti-UK114 monoclonal antibodies selected from the immunoglobulin repertoire of a cancer patient

L33 ANSWER 4 OF 36 HCAPLUS COPYRIGHT 2007 ACS on STN
TI Enhancement of in vitro and in vivo anti-tumor activity of anti-GD2 monoclonal antibody 220-51 against human neuroblastoma by granulocyte-macrophage colony-stimulating factor and granulocyte colony-stimulating factor

L33 ANSWER 5 OF 36 HCAPLUS COPYRIGHT 2007 ACS on STN
TI Anti-GD2 antibody treatment of minimal residual stage 4 neuroblastoma diagnosed at more than 1 year of age

L33 ANSWER 6 OF 36 HCAPLUS COPYRIGHT 2007 ACS on STN

TI Phase I trial of a human-mouse chimeric anti-disialoganglioside monoclonal antibody ch14.18 in patients with refractory neuroblastoma and osteosarcoma

L33 ANSWER 7 OF 36 HCAPLUS COPYRIGHT 2007 ACS on STN
TI Bactericidal monoclonal antibodies that define unique meningococcal B polysaccharide epitopes that do not cross-react with human polysialic acid

L33 ANSWER 8 OF 36 HCAPLUS COPYRIGHT 2007 ACS on STN
TI Localization and characterization of antigenic components of human neuroblastoma cell line SK-N-SH using monoclonal antibodies

L33 ANSWER 9 OF 36 HCAPLUS COPYRIGHT 2007 ACS on STN
TI Additive cytotoxicity of different monoclonal antibody -cobra venom factor conjugates for human neuroblastoma cells

L33 ANSWER 10 OF 36 HCAPLUS COPYRIGHT 2007 ACS on STN
TI Lysis of human tumor cell lines by canine complement plus monoclonal antiganglioside antibodies or natural canine xenoantibodies

L33 ANSWER 11 OF 36 HCAPLUS COPYRIGHT 2007 ACS on STN
TI Complement C1 inhibitor is produced by brain tissue and is cleaved in Alzheimer disease

L33 ANSWER 12 OF 36 HCAPLUS COPYRIGHT 2007 ACS on STN
TI Target cells of cytotoxic T lymphocytes directed to the individual structure proteins of rabies virus

L33 ANSWER 13 OF 36 HCAPLUS COPYRIGHT 2007 ACS on STN
TI Chemotactic activity of substances derived from antibody-loaded tumor cells on granulocytes

L33 ANSWER 14 OF 36 HCAPLUS COPYRIGHT 2007 ACS on STN
TI Genetic engineering and anticancer activities of human anti-ganglioside GM2 antibodies containing mouse heavy and light chain variable regions

L33 ANSWER 15 OF 36 HCAPLUS COPYRIGHT 2007 ACS on STN
TI Immunocytochemical study on internalization of anti-carbohydrate monoclonal antibodies

L33 ANSWER 16 OF 36 HCAPLUS COPYRIGHT 2007 ACS on STN
TI Characterization of antigenic components of human neuroblastoma using monoclonal antibodies

L33 ANSWER 17 OF 36 HCAPLUS COPYRIGHT 2007 ACS on STN
TI SP-40,40 is a constituent of Alzheimer's amyloid

L33 ANSWER 18 OF 36 HCAPLUS COPYRIGHT 2007 ACS on STN
TI Antibodies and autoantigen and methods for diagnosis and treatment of insulin-dependent diabetes mellitus

L33 ANSWER 19 OF 36 HCAPLUS COPYRIGHT 2007 ACS on STN
TI Establishment of anti-human neuroblastoma-selective isotype-switch variants

L33 ANSWER 20 OF 36 HCAPLUS COPYRIGHT 2007 ACS on STN
TI Complement killing of human neuroblastoma cells: a cytotoxic monoclonal antibody and its F(ab)'2-cobra venom factor conjugate are equally cytotoxic

L33 ANSWER 21 OF 36 HCAPLUS COPYRIGHT 2007 ACS on STN
TI Monoclonal antibodies to disialogangliosides: characterization of antibody-mediated cytotoxicity against human melanoma and neuroblastoma cells in vitro

L33 ANSWER 22 OF 36 HCAPLUS COPYRIGHT 2007 ACS on STN
TI Monoclonal antibodies against epitopes on ganglioside GD2 and its lactones. Markers for gliomas and neuroblastomas

L33 ANSWER 23 OF 36 HCAPLUS COPYRIGHT 2007 ACS on STN
TI Functional properties and effect on growth suppression of human neuroblastoma tumors by isotype switch variants of monoclonal antiganglioside GD2 antibody 14.18

L33 ANSWER 24 OF 36 HCAPLUS COPYRIGHT 2007 ACS on STN
TI Monoclonal paratopic molecule directed to human ganglioside GD2 and its use in tumor diagnosis and therapy

L33 ANSWER 25 OF 36 HCAPLUS COPYRIGHT 2007 ACS on STN
TI GM-CSF enhances 3F8 monoclonal antibody-dependent cellular cytotoxicity against human melanoma and neuroblastoma

L33 ANSWER 26 OF 36 HCAPLUS COPYRIGHT 2007 ACS on STN
TI New anti-GD2 monoclonal antibodies produced from gamma-interferon-treated neuroblastoma cells

L33 ANSWER 27 OF 36 HCAPLUS COPYRIGHT 2007 ACS on STN
TI Disialoganglioside GD2 on human neuroblastoma cells: target antigen for monoclonal antibody-mediated cytolysis and suppression of tumor growth

L33 ANSWER 28 OF 36 HCAPLUS COPYRIGHT 2007 ACS on STN
TI A monoclonal anti-neuroblastoma antibody that discriminates between human nonhematopoietic and hematopoietic cell types

L33 ANSWER 29 OF 36 HCAPLUS COPYRIGHT 2007 ACS on STN
TI Monoclonal antibody directed to human ganglioside GD2

L33 ANSWER 30 OF 36 HCAPLUS COPYRIGHT 2007 ACS on STN
TI Hybrid cell line and its use

L33 ANSWER 31 OF 36 HCAPLUS COPYRIGHT 2007 ACS on STN
TI Selection of variant neuroblastoma cell line which has lost cell surface expression of antigen detected by monoclonal antibody PI153/3

L33 ANSWER 32 OF 36 HCAPLUS COPYRIGHT 2007 ACS on STN
TI Protection against 17D yellow fever encephalitis in mice by passive transfer of monoclonal antibodies to the nonstructural glycoprotein gp48 and by active immunization with gp48

L33 ANSWER 33 OF 36 HCAPLUS COPYRIGHT 2007 ACS on STN
TI Monoclonal antibody to small cell carcinoma of human lung

L33 ANSWER 34 OF 36 HCAPLUS COPYRIGHT 2007 ACS on STN
TI Monoclonal antibodies to a glycolipid antigen on human neuroblastoma cells

L33 ANSWER 35 OF 36 HCAPLUS COPYRIGHT 2007 ACS on STN
TI Human monoclonal antibody to tumor-associated ganglioside GD2

L33 ANSWER 36 OF 36 HCAPLUS COPYRIGHT 2007 ACS on STN
TI A membrane glycoprotein from human neuroblastoma cells isolated with the use of a monoclonal antibody

=> d l33 2 4 5 6 20 21 25 ti abs bib
YOU HAVE REQUESTED DATA FROM FILE 'HCAPLUS' - CONTINUE? (Y)/N:y

L33 ANSWER 2 OF 36 HCAPLUS COPYRIGHT 2007 ACS on STN
TI Treatment of neoplastic meningeal xenografts by intraventricular administration of an anti-ganglioside monoclonal antibody, 3F8
AB Leptomeningeal (LM) neoplastic metastases are painful, debilitating and inevitably lethal. Intrathecal (IT) anti-tumor antibodies may have therapeutic potential. We evaluated 3F8, an anti-GD2 murine IgG3 monoclonal antibody (MAb) in the treatment of human melanoma (SKMEL-I) and neuroblastoma (NMB7) xenografts in athymic rats. Both tumors were lysed efficiently in vitro by 3F8 in the presence of rat neutrophils or rat complement. Antibody-dependent cellular cytotoxicity (ADCC) was not augmented by recombinant human GM-CSF (rhGM-CSF), rhG-CSF, recombinant rat MIP-2 (rrMIP-2) or lipopolysaccharide (LPS). In vivo, continuous intraventricular administration of 3F8 and LPS prevented tumor engraftment, retarded tumor growth and eradicated 3-day-old established xenografts whereas 3F8 alone, LPS alone or F(ab)'2 plus LPS had no or only marginal effects. Tumor establishment in brain was completely prevented in 36% of animals implanted with SKMEL-I and 65% of animals implanted with NMB7. Twenty percent of established xenografts around the brain were eradicated but all animals had persistent tumor in the lumbosacral meninges despite treatment. Continuous intraventricular infusion of LPS produced a variable polymorphonuclear (PMN) pleocytosis that was dose-dependent. Continuous intraventricular infusion of 3F8 produced immunohistochem. detectable attachment to 86% of persistent brain deposits of tumor but < 1% of spinal lumbosacral deposits. We conclude that regional therapy with anti-GD2 MAb could target neutrophils to inhibit LM tumor growth. However, optimal activation and mobilization of neutrophils into the cerebrospinal fluid (CSF) and improved penetration of MAb to tumor sites remain critical variables.
AN 1999:506942 HCAPLUS <<LOGINID::20070726>>
DN 132:48758
TI Treatment of neoplastic meningeal xenografts by intraventricular administration of an anti-ganglioside monoclonal antibody, 3F8
AU Bergman, Ira; Barmada, Mamdouha A.; Heller, Glenn; Griffin, Judith A.; Cheung, Nai-Kong V.
CS Departments of Pediatrics and Neurology, University of Pittsburgh Medical Center, Pittsburgh, PA, USA
SO International Journal of Cancer (1999), 82(4), 538-548
CODEN: IJCNAW; ISSN: 0020-7136
PB Wiley-Liss, Inc.
DT Journal
LA English
RE.CNT 41 THERE ARE 41 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L33 ANSWER 4 OF 36 HCAPLUS COPYRIGHT 2007 ACS on STN
TI Enhancement of in vitro and in vivo anti-tumor activity of anti-GD2 monoclonal antibody 220-51 against human neuroblastoma by granulocyte-macrophage colony-stimulating factor and granulocyte colony-stimulating factor
AB We have evaluated the anti-tumor effect of anti-GD2 mouse monoclonal antibody (mAb) 220-51 against human neuroblastoma cell line TGW in vitro and in vivo. The mAb 220-51 was able to mediate complement-dependent cytotoxicity (CDC) and antibody-dependent cellular cytotoxicity (ADCC) using human effector cells. In the presence of recombinant human granulocyte-macrophage colony-stimulating factor (GM-CSF), granulocyte ADCC was significantly

augmented in vitro. When mAb 220-51 was administered to tumor-bearing nude mice, tumor growth was significantly inhibited as compared with untreated controls. Administration of recombinant murine GM-CSF in combination with mAb 220-51 significantly enhanced the anti-tumor effect of mAb in vivo. Recombinant human granulocyte colony-stimulating factor (G-CSF) combined with mAb 220-51 was also able to enhance it, although granulocyte ADCC was not affected by the presence of recombinant human G-CSF in vitro. Moreover, GM-CSF and G-CSF work additively to enhance the anti-tumor effect of mAb 220-51 in vivo. The GM-CSF and G-CSF may have a clin. potency in immunotherapy with anti-GD2 mAb for the treatment of neuroblastoma.

AN 1998:691201 HCAPLUS <<LOGINID::20070726>>
DN 130:94183
TI Enhancement of in vitro and in vivo anti-tumor activity of anti-GD2 monoclonal antibody 220-51 against human neuroblastoma by granulocyte-macrophage colony-stimulating factor and granulocyte colony-stimulating factor
AU Fukuda, Minoru; Horibe, Keizo; Furukawa, Koichi
CS Department of Pediatrics, Nagoya University School of Medicine, Nagoya, 466-8550, Japan
SO International Journal of Molecular Medicine (1998), 2(4), 471-475
CODEN: IJMMFG; ISSN: 1107-3756
PB International Journal of Molecular Medicine
DT Journal
LA English
RE.CNT 28 THERE ARE 28 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L33 ANSWER 5 OF 36 HCAPLUS COPYRIGHT 2007 ACS on STN
TI Anti-GD2 antibody treatment of minimal residual stage 4 neuroblastoma diagnosed at more than 1 year of age
AB The purpose of this trial was to eradicate minimal residual disease with anti-GD2 monoclonal antibody 3F8 in stage 4 neuroblastoma (NB) diagnosed at more than 1 yr of age. Thirty-four patients were treated with 3F8 at the end of chemotherapy. Most had either bone marrow (n = 31) or distant bony metastases (n = 29). Thirteen patients were treated at second or subsequent remission (group I) and 12 patients in this group had a history of progressive/persistent disease after bone marrow transplantation (BMT); 21 patients were treated in first remission following N6 chemotherapy (group II). Before 3F8 treatment, 23 patients were in complete remission CR, eight in very good partial remission (VGPR), one in partial remission (PR), and two had microscopic foci in marrow. Twenty-five had evidence of NB by at least one measurement of occult/minimal tumor (iodine 131[131I]-3F8 imaging, marrow immunocytol., or marrow reverse-transcriptase polymerase chain reaction [RT-PCR]). Acute self-limited toxicities of 3F8 treatment were severe pain, fever, urticaria, and reversible decreases in blood counts and serum complement levels. There was evidence of response by immunocytol. (six of nine), by GAGE RT-PCR (seven of 12), and by 131I-3F8 scans (six of six). Fourteen patients are alive and 13 (age 1.8 to 7.4 yr at diagnosis) are progression-free (40 to 130 mo from the initiation of 3F8 treatment) without further systemic therapy, none with late neurol. complications. A transient anti-mouse response or the completion of four 3F8 cycles was associated with significantly better survival. Despite high-risk nature of stage 4 NB, long-term remission without autologous (A)BMT can be achieved with 3F8 treatment. Its side effects were short-lived and manageable. The potential benefits of 3F8 in consolidating remission warrant further investigations.

AN 1998:610625 HCAPLUS <<LOGINID::20070726>>
DN 130:23877
TI Anti-GD2 antibody treatment of minimal residual stage 4 neuroblastoma diagnosed at more than 1 year of age
AU Cheung, Nai-Kong V.; Kushner, Brian H.; Cheung, Irene Y.; Kramer, Kim;

Canete, Adela; Gerald, William; Bonilla, Mary Ann; Finn, Ronald; Yeh, Samuel J.; Larson, Steven M.
CS Departments of Pediatrics, Pathology, and Medical Imaging, Memorial Sloan-Kettering Cancer Center, New York, NY, 10021, USA
SO Journal of Clinical Oncology (1998), 16(9), 3053-3060
CODEN: JCONDN; ISSN: 0732-183X
PB W. B. Saunders Co.
DT Journal
LA English
RE.CNT 37 THERE ARE 37 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L33 ANSWER 6 OF.36 HCAPLUS COPYRIGHT 2007 ACS on STN
TI Phase I trial of a human-mouse chimeric anti-disialoganglioside monoclonal antibody ch14.18 in patients with refractory neuroblastoma and osteosarcoma
AB To evaluate the toxicity, immunogenicity, and pharmacokinetics of a human-mouse chimeric monoclonal antibody (mAb) ch14.18 directed against disialoganglioside (GD2) and to obtain preliminary information on its clin. efficacy, we conducted a phase I trial in 10 patients with refractory neuroblastoma and one patient with osteosarcoma. Eleven patients were entered onto this phase I trial. They received 20 courses of mAb ch14.18 at dose levels of 10, 20, 50, 100, and 200 mg/m2. Dose escalation was performed in cohorts of three patients; intrapatient dose escalation was also permitted. The most prevalent toxicities were pain, tachycardia, hypertension, fever, and urticaria. Most of these toxicities were dose-dependent and rarely noted at dosages of 20 mg/m2 and less. Although the maximum-tolerated dose was not reached in this study, clin. responses were observed These included one partial (PR) and four mixed responses (MRs) and one stable disease (SD) among 10 assessable patients. Biol. activity of ch14.18 in vivo was shown by binding of ch14.18 to tumor cells and complement-dependent cytotoxicity of post-treatment sera against tumor target cells. An anti-ch14.18 immune response was detectable in seven of 10 patients studied. In summary, with the dose schedule used, ch14.18 appears to be clin. safe and effective, and repeated mAb administration was not associated with increased toxicities. Further clin. trials of mAb ch14.18 in patients with neuroblastoma are warranted.
AN 1998:409136 HCAPLUS <<LOGINID::20070726>>
DN 129:188243
TI Phase I trial of a human-mouse chimeric anti-disialoganglioside monoclonal antibody ch14.18 in patients with refractory neuroblastoma and osteosarcoma
AU Yu, Alice L.; Uttenreuther-Fischer, Martina M.; Huang, Chiun-Sheng; Tsui, Cynthia C.; Gillies, Steven D.; Reisfeld, Ralph A.; Kung, Faith H.
CS Department of Pediatrics, Division of Pediatric Hematology/Oncology, University of California San Diego, San Diego, CA, USA
SO Journal of Clinical Oncology (1998), 16(6), 2169-2180
CODEN: JCONDN; ISSN: 0732-183X
PB W. B. Saunders Co.
DT Journal
LA English
RE.CNT 52 THERE ARE 52 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L33 ANSWER 20 OF 36 HCAPLUS COPYRIGHT 2007 ACS on STN
TI Complement killing of human neuroblastoma cells: a cytotoxic monoclonal antibody and its F(ab)'2-cobra venom factor conjugate are equally cytotoxic
AB Only a few monoclonal antibodies mediate complement lysis of tumor cells, but for several antibodies it has been demonstrated that a complement-activating function can be introduced by covalent coupling of cobra venom factor (CVF), a non-toxic glycoprotein which is a structural and functional homolog of human complement component

C3. In this study the authors compared the efficacy of complement killing of human neuroblastoma cells by the complement -activating monoclonal antibody 3F8 directed against the GD2 ganglioside antigen with that of its F(ab')2-CVF conjugate. At equal nos. bound per cell the 3F8 antibody and the 3F8 F(ab')2-CVF conjugate were found to be equally cytotoxic in the presence of complement from several species including human. Maximal killing reached up to 98%. The kinetics of killing and the bivalent metal requirement confirmed that the cytotoxic activity of the 3F8 antibody is mediated via the classical pathway and that of the 3F8 F(ab')2-CVF conjugate via the alternative pathway. To achieve a comparable degree of killing, an approx. eight-fold higher concentration of the 3F8 F(ab')2-CVF conjugate was required which appears to be a consequence of the approx. eight-fold lower binding activity of the 3F8 F(ab')2-CVF conjugate to the intact 3F8 antibody. These data suggest that the coupling of CVF to non-cytotoxic antibodies allows the generation of conjugates with a cytotoxic activity similar to that of inherently cytotoxic antibodies.

AN 1990:624272 HCAPLUS <<LOGINID::20070726>>
DN 113:224272
TI Complement killing of human neuroblastoma cells: a cytotoxic monoclonal antibody and its F(ab)'2-cobra venom factor conjugate are equally cytotoxic
AU Juhl, Hartmut; Petrella, Eugene C.; Cheung, Nai Kong V.; Bredehorst, Reinhard; Vogel, Carl Wilhelm
CS Vincent T. Lombardi Cancer Cent., Georgetown Univ., Washington, DC, 20007, USA
SO Molecular Immunology (1990), 27(10), 957-64
CODEN: MOIMD5; ISSN: 0161-5890
DT Journal
LA English

L33 ANSWER 21 OF 36 HCAPLUS COPYRIGHT 2007 ACS on STN
TI Monoclonal antibodies to disialogangliosides: characterization of antibody-mediated cytotoxicity against human melanoma and neuroblastoma cells in vitro
AB It was previously reported the binding specificities of two anti-ganglioside GD2 murine monoclonal antibodies (MAbs), A1-425 and A1-267, both of which are of IgG3 isotype. A1-425 reacts specifically with ganglioside GD2, whereas A1-267 binds preferentially to GD2 but also reacts with GD3 (Tai, T., et al 1988). In this paper, they were used for comparative analyses of antibody-mediated cytotoxicity, i.e., antibody-dependent cellular cytotoxicity (ADCC) and complement -dependent cytotoxicity (CDC) against human melanoma and neuroblastoma cell lines. Melanoma cells were found to contain GD2 and/or GD3, whereas neuroblastoma cells expressed only GD2. Both antibodies induced high levels of ADCC and CDC to GD2/GD3-pos. cells with human peripheral large granular lymphocytes (LGL) as effector cells and in the presence of human serum, resp. Antigen-antibody complexes composed of GD2 and A1-425 showed higher binding levels to LGL than complexes of GD2 and A1-267. In contrast, free MAb mols. gave min. binding to LGL. An anti-human Fc-receptors (III) MAb specifically inhibited both the binding of the antigen-antibody complex to LGL and the ADCC by the MAbs with LGL. These findings demonstrate that MAbs having high binding levels to Fc-receptors (III), as well as having specificities towards multiple ganglioside antigens, possess the strongest cytotoxicity against human tumor cells in ADCC.

AN 1990:476175 HCAPLUS <<LOGINID::20070726>>
DN 113:76175
TI Monoclonal antibodies to disialogangliosides: characterization of antibody-mediated cytotoxicity against human melanoma and neuroblastoma cells in vitro
AU Kawashima, Ikuo; Tada, Nobuhiko; Fujimori, Takao; Tai, Tadashi
CS Dep. Tumor Immunol., Tokyo Metrop. Inst. Med. Sci., Tokyo, 113, Japan
SO Journal of Biochemistry (Tokyo, Japan) (1990), 108(1), 109-15

CODEN: JOBIAO; ISSN: 0021-924X
DT Journal
LA English

L33 ANSWER 25 OF 36 HCAPLUS COPYRIGHT 2007 ACS on STN
TI GM-CSF enhances 3F8 monoclonal antibody-dependent cellular cytotoxicity against human melanoma and neuroblastoma
AB Antibody 3F8 is a murine monoclonal IgG3 antibody specific for the tumor-associated antigen ganglioside GD2. Previous in vitro studies suggest that tumor regressions observed in a phase I clin. trial of 3F8 may be attributable to complement activation by 3F8 and to 3F8-dependent cellular cytotoxicity (ADCC) with lymphocytes. Here, it is shown that 3F8 mediated ADCC of GD2-pos. tumor targets (melanoma and neuroblastoma) with human granulocytes and that recombinant human granulocyte-macrophage colony-stimulating factor (GM-CSF) enhanced this phenomenon. Cytotoxicity required binding of 3F8 to the low-affinity Fc receptor type III (CD16) on the granulocytes and was poor with tumor-binding monoclonal antibodies of other Ig (i.e., non-IgG3) subclasses. Nonoxidative mechanisms may be important for ADCC since 3F8 mediated ADCC with granulocytes from 2 children with chronic granulomatous disease; this cytotoxicity was also enhanced by GM-CSF. Since GM-CSF induces a neutrophilia in patients, this cytokine may have the potential of amplifying 3F8 antitumor activity in patients by increasing effector cell nos. and by priming granulocytes for greater cytotoxicity.
AN 1989:405582 HCAPLUS <<LOGINID::20070726>>
DN 111:5582
TI GM-CSF enhances 3F8 monoclonal antibody-dependent cellular cytotoxicity against human melanoma and neuroblastoma
AU Kushner, Brian H.; Cheung, Nai Kong V.
CS Dep. Pediatr., Mem. Sloan-Kettering Cancer Cent., New York, NY, 10021, USA
SO Blood (1989), 73(7), 1936-41
CODEN: BLOOAW; ISSN: 0006-4971
DT Journal
LA English

=> d l34 1-9 ti
YOU HAVE REQUESTED DATA FROM FILE 'HCAPLUS' - CONTINUE? (Y)/N:y

L34 ANSWER 1 OF 9 HCAPLUS COPYRIGHT 2007 ACS on STN
TI Overview of the clinical development of rituximab: First monoclonal antibody approved for the treatment of lymphoma

L34 ANSWER 2 OF 9 HCAPLUS COPYRIGHT 2007 ACS on STN
TI Antibody-targeted therapy for low-grade lymphoma

L34 ANSWER 3 OF 9 HCAPLUS COPYRIGHT 2007 ACS on STN
TI FC-2.15, a monoclonal antibody active against human breast cancer, specifically recognizes Lewisx hapten

L34 ANSWER 4 OF 9 HCAPLUS COPYRIGHT 2007 ACS on STN
TI IDEC-C2B8 (rituximab) anti-CD20 monoclonal antibody therapy in patients with relapsed low-grade non-Hodgkin's lymphoma

L34 ANSWER 5 OF 9 HCAPLUS COPYRIGHT 2007 ACS on STN
TI Therapeutic and diagnostic methods using leukocyte surface antigens

L34 ANSWER 6 OF 9 HCAPLUS COPYRIGHT 2007 ACS on STN
TI Immunological purging of tumor cells from bone marrow using microspheres and monoclonal antibodies

L34 ANSWER 7 OF 9 HCAPLUS COPYRIGHT 2007 ACS on STN
TI Production of a monoclonal antibody (IND.64) identifying a cell cycle-associated antigen using spleen cells from nude mice bearing Ichikawa tumor

L34 ANSWER 8 OF 9 HCAPLUS COPYRIGHT 2007 ACS on STN
TI A novel human leukocyte surface membrane antigen defined by murine monoclonal antibody

L34 ANSWER 9 OF 9 HCAPLUS COPYRIGHT 2007 ACS on STN
TI Effects of methotrexate on natural killer cell activity in vitro and in vivo

=> d l34 1 2 3 4 5 6 ti abs bib
YOU HAVE REQUESTED DATA FROM FILE 'HCAPLUS' - CONTINUE? (Y)/N:y

L34 ANSWER 1 OF 9 HCAPLUS COPYRIGHT 2007 ACS on STN
TI Overview of the clinical development of rituximab: First monoclonal antibody approved for the treatment of lymphoma
AB A review with 18 refs. Rituximab (Rituxan; IDEC Pharmaceuticals, San Diego, CA, and Genentech, Inc, San Francisco, CA) is a genetically engineered monoclonal antibody for the treatment of non-Hodgkin's lymphoma. This chimeric mouse/human, Ig GI kappa anti-CD20 antibody mediates complement-dependent cell lysis and antibody-dependent cellular cytotoxicity. It also has been shown to sensitize chemoresistant human lymphoma cell lines and to induce apoptosis. It was approved by the Food and Drug Administration on Nov. 26, 1997, for the indication of relapsed or refractory, CD-20 pos., B-cell, low-grade or follicular non-Hodgkin's lymphoma . Rituximab is the first monoclonal antibody approved for the treatment of cancer and the first single agent approved specifically for therapy of a lymphoma. The recommended dose is rituximab 375 mg/m2 i.v. weekly +4 infusions. Treatment is well tolerated and outpatient therapy is feasible. Adverse events are mostly grades 1 and 2, occurring primarily with the first infusion. In a phase II single-agent clin. trial, the overall response rate was 50%, with a median time to progression in responders of 10.2 mo. In a larger multicenter trial involving 166 patients, the overall response rate was 48% with 6% complete and 42% partial responses. Median time to progression for responders was 13.2 mo and median duration of response was 11.6 mo. A 40% response rate has been observed on re-treatment with rituximab. Activity also has been seen in patients with bulky disease. Combination studies have been performed with interferon, cyclophosphamide/doxorubicin/vincristine/predni sone, and radioimmunotherapy. Rituximab, the first monoclonal antibody approved for the treatment of cancer, is safe and effective in treating patients with relapsed or refractory, CD-20 pos., B-cell, low-grade or follicular non-Hodgkin's lymphoma .
AN 1999:775052 HCAPLUS <<LOGINID::20070726>>
DN 131:350015
TI Overview of the clinical development of rituximab: First monoclonal antibody approved for the treatment of lymphoma
AU Grillo-Lopez, Antonio J.; White, Christine A.; Varns, Chet; Shen, David; Wei, Alice; McClure, Anne; Dallaire, Brian K.
CS IDEC Pharmaceuticals Corporation, San Diego, CA, 92121, USA
SO Seminars in Oncology (1999), 26(5, Suppl. 14), 66-73
CODEN: SOLGAV; ISSN: 0093-7754
PB W. B. Saunders Co.

DT Journal; General Review
LA English
RE.CNT 18 THERE ARE 18 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L34 ANSWER 2 OF 9 HCAPLUS COPYRIGHT 2007 ACS on STN
TI Antibody-targeted therapy for low-grade lymphoma
AB A review with 29 refs. Monoclonal antibodies (MoAbs) have now become a successful treatment for selected patients with non-Hodgkin's lymphoma (NHL). Antibody targets most commonly used for the treatment of B-cell NHL include CD20, CD19, and CD22. Unconjugated MoAbs are cytotoxic by several mechanisms, including complement -dependent cytotoxicity (CDC), antibody-dependent cell-mediated cytotoxicity (ADCC), and signal transduction leading to apoptosis. In an attempt to augment the effectiveness of naked antibody prepns., various radioconjugates, immunotoxins, chemotherapeutic agents, or immune-modifiers have been attached to the antibodies. The immunotoxin tested most extensively in clin. trials is B4-blocked ricin (anti-CD19 with a partially blocked ricin toxin). The use of radioimmunoconjugates to augment the effectiveness of unlabeled antibodies has been one of the most popular strategies. Antibodies against these targets have now been chelated with radioconjugates such as 131I or 90Y and tested in recent clin. trials. Radioimmunotherapy has the theor. advantage over naked antibody therapy or immunotoxin therapy in that the MoAb conjugated with a radioisotope can have a "cross-fire" effect such that antigen-neg. tumor cells adjacent to those expressing the target antigen may also be killed. This may enhance the likelihood of tumor sterilization even in fairly bulky disease. Future studies will focus on testing these antibodies in larger patient populations, sequentially or in combination, and on combining MoAb therapy with standard- or high-dose chemotherapy and hematopoietic stem-cell transplantation.
AN 1999:710549 HCAPLUS <<LOGINID::20070726>>
DN 132:220894
TI Antibody-targeted therapy for low-grade lymphoma
AU Vose, Julie M.
CS Department of Internal Medicine, University of Nebraska Medical Center, Omaha, NE, 68198-3332, USA
SO Seminars in Hematology (1999), 36(4, Suppl. 6), 15-20
CODEN: SEHEA3; ISSN: 0037-1963
PB W. B. Saunders Co.
DT Journal; General Review
LA English
RE.CNT 29 THERE ARE 29 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L34 ANSWER 3 OF 9 HCAPLUS COPYRIGHT 2007 ACS on STN
TI FC-2.15, a monoclonal antibody active against human breast cancer, specifically recognizes Lewisx hapten
AB FC-2.15 is a murine IgM monoclonal antibody that recognizes breast and colon human carcinomas, chronic myeloid leukemias, Sternberg cells of Hodgkin's lymphoma and some normal cells, such as peripheral polymorphonuclear granulocytes. It has been previously demonstrated that FC-2.15 recognizes the carbohydrate moiety of different glycoproteins. FC-2.15 is able to mediate the in vitro lysis of Ag-2.15+ cells by human complement. In a phase I clin. trial, FC-2.15 induced antitumor responses and reversible neutropenia was its main toxicity. In this work, anal. of epitope specificity has demonstrated that FC-2.15 specifically recognizes terminally exposed Lewisx trisaccharide but not sialyl-Lewisx, Lewisa, trifucosylated Lewisy, blood-group antigens A and B, globo H and gangliosides. In polymorphonuclear granulocytes (PMN), myeloid leukemic cells and colon carcinoma T84 cells, Lewisx was found to be almost exclusively N-linked to the protein core, whereas in breast carcinoma MCF-7 cells, Lewisx appeared to be mostly O-linked. Treatment with neuraminidase increased detection

by FC-2.15 in normal PMN, myeloid leukemia cells and T84 cells but not in MCF-7 cells.
AN 1998:129066 HCAPLUS <<LOGINID::20070726>>
DN 128:203919
TI FC-2.15, a monoclonal antibody active against human breast cancer, specifically recognizes Lewisx hapten
AU Capurro, Mariana; Bover, Laura; Portela, Paula; Livingston, Philip; Mordoh, Jose
CS Instituto de Investigaciones Bioquimicas "Fundacion Campomar", Buenos Aires, 1405, Argent.
SO Cancer Immunology Immunotherapy (1998), 45(6), 334-339
CODEN: CIIMDN; ISSN: 0340-7004
PB Springer-Verlag
DT Journal
LA English
RE.CNT 20 THERE ARE 20 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L34 ANSWER 4 OF 9 HCAPLUS COPYRIGHT 2007 ACS on STN
TI IDEC-C2B8 (rituximab) anti-CD20 monoclonal antibody therapy in patients with relapsed low-grade non-Hodgkin's lymphoma
AB IDEC-C2B8 is a chimeric monoclonal antibody (MoAb) directed against the B-cell-specific antigen CD20 expressed on non-Hodgkin's lymphomas (NHL). The MoAb mediates complement and antibody-dependent cell-mediated cytotoxicity and has direct antiproliferative effects against malignant B-cell lines in vitro. Phase I trials of single doses up to 500 mg/m2 and 4 weekly doses of 375 mg/m2 showed clin. responses with no dose-limiting toxicity. We conducted a phase II, multicenter study evaluating four weekly infusions of 375 mg/m2 IDEC-C2B8 in patients with relapsed low-grade or follicular NHL (Working Formulation groups A-D). Patients were monitored for adverse events, antibody pharmacokinetics, and clin. response. Thirty-seven patients with a median age of 58 yr (range, 29 to 81 yr) were treated. All patients had relapsed after chemotherapy (median of 2 prior regimens) and 54% had failed aggressive chemotherapy. Infusional side effects (grade 1-2) consisting of mild fever, chills, respiratory symptoms, and occasionally hypotension were observed mostly with the initial antibody infusion and were rare with subsequent doses. Peripheral blood B-cell depletion occurred rapidly, with recovery beginning 6 mo posttreatment. There were no significant changes in mean IgG levels and infections were not increased over what would be expected in this population. Clin. remissions were observed in 17 patients (3 complete remissions and 14 partial remissions), yielding an intent to treat response rate of 46%. The onset of these tumor responses was as soon as 1 mo posttreatment and reached a maximum by 4 mo posttreatment. In the 17 responders, the median time to progression was 10.2 mo (5 patients exceeding 20 mo). Likelihood of tumor response was associated with a follicular histol., with the ability to sustain a high serum level of antibody after the first infusion, and with a longer duration of remission to prior chemotherapy. One patient developed a detectable but not quantifiable immune response to the antibody that had no clin. significance. IDEC-C2B8 in a dose of 375 mg/m2 weekly for 4 wk has antitumor activity in patients with relapsed low-grade or follicular NHL. Results with this brief, outpatient treatment compare favorably with results with standard chemotherapy, and IDEC-C2B8 has a better safety profile. Further studies evaluating IDEC-C2B8 in other types of lymphoma either alone or combined with chemotherapy are warranted.
AN 1997:607923 HCAPLUS <<LOGINID::20070726>>
DN 127:276918
TI IDEC-C2B8 (rituximab) anti-CD20 monoclonal antibody therapy in patients with relapsed low-grade non-Hodgkin's lymphoma
AU Maloney, David G.; Grillo-Lopez, Antonio J.; White, Christine A.; Bodkin, David; Schilder, Russell J.; Neidhart, James A.; Janakiraman, Nalini;

Foon, Kenneth A.; Liles, Tina-Marie; Dallaire, Brian K.; Wey, Ken; Royston, Ivor; Davis, Thomas; Levy, Ronald

CS Department of Medicine, Division of Oncology, Stanford University, Stanford, CA, USA

SO Blood (1997), 90(6), 2188-2195
CODEN: BLOOAW; ISSN: 0006-4971

PB Saunders

DT Journal

LA English

RE.CNT 35 THERE ARE 35 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L34 ANSWER 5 OF 9 HCAPLUS COPYRIGHT 2007 ACS on STN

TI Therapeutic and diagnostic methods using leukocyte surface antigens

AB Measurement of soluble leukocyte surface markers, soluble T-cell growth factor receptors, soluble complement receptors, soluble T-cell differentiation antigens, or related soluble mols. or fragments, particularly soluble CD4, CD8, and CD35, are useful in the diagnosis and therapy of diseases and disorders. A polyclonal sandwich EIA is provided for the detection and/or measurement of soluble CD35. The invention further relates to measurement of total leukocyte markers or fragments (including those present in membrane and intracellular compartments and extracellular soluble compartments) in disease detection and diagnosis. Measurements of a total leukocyte marker can be used to determine the approx. amount in a body fluid sample of leukocytes pos. for the leukocyte marker. Soluble CD35 was detected in serum of patients with lupus erythematosus, renal transplant, osteosarcoma, Hodgkin's disease, and leukemia.

AN 1995:712290 HCAPLUS <<LOGINID::20070726>>

DN 123:107265

TI Therapeutic and diagnostic methods using leukocyte surface antigens

IN Rittershaus, Charles W.; Tian, Wei Tao; Kung, Patrick C.

PA T Cell Diagnostics, Inc., USA

SO U.S., 52 pp. Cont.-in-part of U.S.5,292,638.
CODEN: USXXAM

DT Patent

LA English

FAN.CNT 6

	PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI	US 5426029	A	19950620	US 1990-610494	19901107 <--
	US 5006459	A	19910409	US 1987-20819	19870302 <--
	EP 699907	A2	19960306	EP 1995-116258	19870303 <--
	EP 699907	A3	19960828		
	R: AT, BE, CH, DE, FR, GB, IT, LI, LU, NL, SE				
	CA 1283045	C	19910416	CA 1987-533323	19870330 <--
	US 5292636	A	19940308	US 1989-434398	19891109 <--
	CA 2095626	A1	19920508	CA 1991-2095626	19911101 <--
	WO 9208981	A1	19920529	WO 1991-US8085	19911101 <--
	W: AU, CA, JP, KR, US				
	RW: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LU, NL, SE				
	AU 9189465	A	19920611	AU 1991-89465	19911101 <--
	AU 671072	B2	19960815		
	EP 556272	A1	19930825	EP 1991-920340	19911101 <--
	EP 556272	B1	19990210		
	R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE				
	JP 06505332	T	19940616	JP 1991-500154	19911101 <--
	AT 176729	T	19990215	AT 1991-920340	19911101 <--
	ES 2129415	T3	19990616	ES 1991-920340	19911101 <--
	WO 9208980	A1	19920529	WO 1991-US8330	19911107 <--
	W: AU, CA, JP, KR, US				
	RW: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LU, NL, SE				
	AU 9189563	A	19920611	AU 1991-89563	19911107 <--
	US 5811525	A	19980922	US 1995-432322	19950501 <--
PRAI	US 1986-846230	B2	19860331 <--		

US 1987-20819	A2	19870302	<--
US 1988-254551	B2	19881006	<--
US 1989-434398	A2	19891109	<--
EP 1987-901965	A3	19870303	<--
US 1990-610494	A	19901107	<--
WO 1991-US8085	A	19911101	<--
WO 1991-US8330	A	19911107	<--
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L34 ANSWER 6 OF 9 HCAPLUS COPYRIGHT 2007 ACS on STN

TI Immunological purging of tumor cells from bone marrow using microspheres and monoclonal antibodies

AB A method is described for the immunol. purging of tumor cells from the bone marrow of a patient having B-cell lymphoma. The purged bone marrow may be used for therapeutic autologous bone marrow transplantation. Microspheres and a plurality of anti-B-cell monoclonal antibodies (MAbs) are used to remove tumor cells (e.g. non-Hodgkin's B-cell lymphoma cells) from bone marrow, without removal or lysis of nontumor cells and without the use of complement, to a level not detectable by PCR assay. The microspheres are especially Ig-coated magnetic microspheres 0.1-5 μm in size. The marrow may be treated with MAbs and microspheres in a specified sequence, or the MAbs may be bound or conjugated to the microspheres. The MAbs are directed especially to antigens B5, CD10, CD19, and/or CD20.

AN 1994:433160 HCAPLUS <<LOGINID::20070726>>

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TI Immunological purging of tumor cells from bone marrow using microspheres and monoclonal antibodies

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SO PCT Int. Appl., 66 pp.
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DT Patent

LA English

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	PATENT NO.	KIND	DATE	APPLICATION NO.	DATE	
PI	WO 9408621	A1	19940428	WO 1993-US9891	19931018	<--
	W: AU, CA, JP					
	RW: AT, BE, CH, DE, DK, ES, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE					
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